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TOMOGRAPHY OF THE LARYNX*

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MY INTRODUCTION to this subject came through Dr. Felix Leborgne[1], of Montevideo, Uruguay, who visited this country in 1938 and brought with him many interesting studies on laryngeal tumors made with a simple device and technique which he had perfected. I believe that to him goes credit for the pioneer work in laryngeal tomography.

It is not the purpose of this communication to discuss the relative merits of the various types of apparatus that have been developed for body section roentgenography. Neither do we hold any controversy with those who prefer the terms such as laminagraphy, stratigraphy and planigraphy as a more appropriate nomenclature.

The apparatus which we use has the virtues of being very simple, inexpensive and easy to operate, and gives us information that heretofore has been within the realm of conjecture.

APPARATUS AND TECHNIQUE

The apparatus (Figs. 1 and 2) consists essentially of three parts. Employing the principles of a simple lever with a fulcrum in the center, the fulcrum assembly, *c*, *d*, is attached in the center of the roentgenographic table to the compression band side rail. The actual fulcrum itself, *c*, moves on a pivot which is adjustable in a calibrated vertical slot. The calibration is expressed in terms of centimeters from the table top. The lever is attached to the supporting structure of the tube and passes through a sleeve in the pivoted fulcrum at *c* and then passes through another pivoted sleeve at *a*. This last named pivoted sleeve at *a* is

FIG. 1. Apparatus. *A*, position of tube at beginning of exposure; *B*, position of tube at end of exposure. *a*, position of Potter-Bucky at beginning of exposure; *b*, position of Potter-Bucky at end of exposure; *c*, adjustable fulcrum; *d*, support for fulcrum.

* From the Radiologic Clinic of Drs. Groover, Christie and Merritt. Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

attached to the center of the Potter-Bucky diaphragm.

During the course of the exposure the tube stand is manually propelled from *A* to *B* and the Potter-Bucky diaphragm moves *a* to *b*. The tube support is locked to the tube stand and the lever slides through the sleeve at *c* and *a* so that during the exposure the tube moves parallel to the table top and to the Potter-Bucky diaphragm.

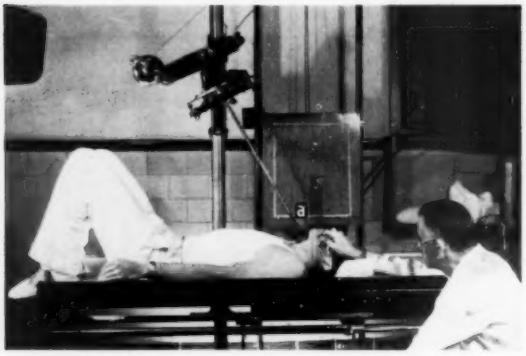

Fig. 2. Position of patient. *a*, support for fulcrum. Note: technician measuring distance for desired planes.

The patient is placed (Fig. 2) in the supine position with the thyroid cartilage lying directly opposite the vertical support of the fulcrum *a*. It is very important that the patient be in the center of the table and the sagittal plane of the head lie exactly perpendicular to the table top. The chin is slightly elevated in order to prevent superimposition of the mandible upon the larynx, but not too much, or there may be superimposition of the base of the skull. It is desirable to approximate the vertical axis of the glottis as nearly horizontal to the table top as possible. The distance from the table top to the anterior border of the thyroid cartilage is then measured and sections are roentgenographed at this level and three subsequent levels 1 cm. apart below. We thus have four exposures of the larynx in the coronal plane at different levels, at least one of which—and sometimes two—is of satisfactory quality.

The technical factors that we employ with this particular apparatus are: 34 inch focal-film distance in the vertical plane; the exposure time is 1½ sec.; 100 ma., and the kilovoltage varying from 50 to 60 kv. (peak), depending upon the thickness of the neck. The tube stand travels the entire length of the table during the exposure. The patient phonates the vowel "e" forcibly while the exposure is being made, unless it is desirable to demonstrate the degree of mobility of the laryngeal structures, and then films are made both with and without phonation. It is desirable to utilize the longest focal-film distance possible in order to secure the minimum of magnified distortion.

We have attempted on several occasions to make lateral tomograms of the larynx but due to the asymmetry of the organ in the sagittal plane they are of less value than the routine roentgen study.

NORMAL LARYNX

In order to properly evaluate the finding on laryngeal tomograms it is necessary to have a thorough understanding of the anatomy and physiology of the larynx as seen with the laryngeal mirror and in the lateral soft tissue roentgenogram. The roentgenologist should be thoroughly adept in the use of the laryngeal mirror and be able to properly correlate the history with the visual examination, the soft tissue roentgenogram and the tomogram. All of these approaches are parts of a picture that must fit together perfectly before a correct diagnosis can be made.

In Figure 3, *A* represents the normal larynx as seen in the laryngeal mirror.* *B* is

*Length of arrow in diagrams indicates relative mobility of the structure.

Fig. 3. Normal larynx. *A*, mirror view; *B*, tomogram during phonation; *C*, lateral view; *D*, tomogram during phonation; *E*, tomogram during phonation; *F*, tomogram without phonation. *a*, epiglottis; *b*, true cord; *c*, false cord; *d*, arytenoid cartilage; *e*, cricoid cartilage; *f*, hyoid bone; *g*, pyriform sinus; *h*, aryepiglottic fold; *i*, ventricular cavity (sinus of Morgagni); *j*, trachea; *k*, laryngeal vestibule; *l*, inferior cornua of thyroid cartilage; *m*, laryngeal face of epiglottis.

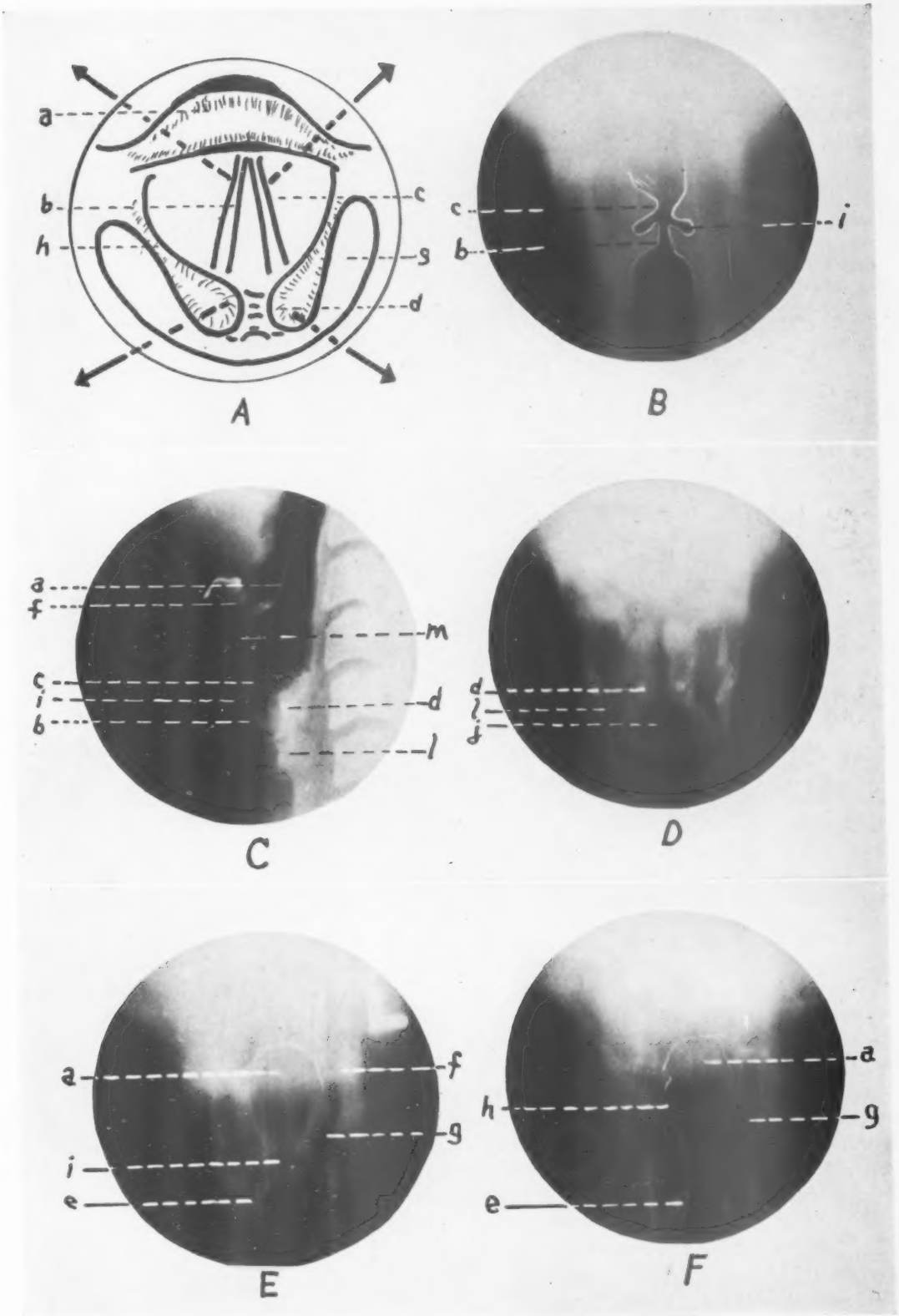

Fig. 3. (*See opposite page for legend.*)

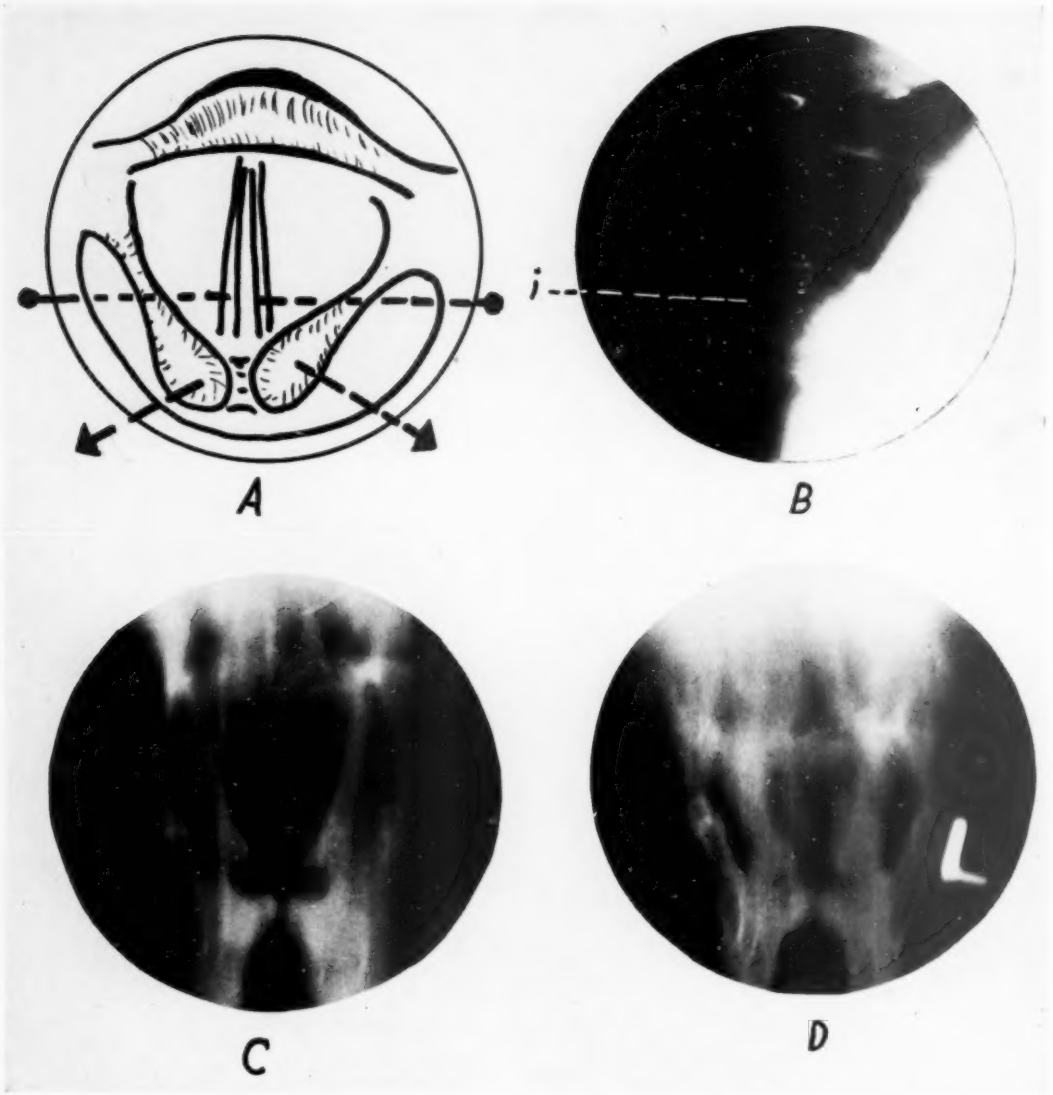

Fig. 4. Case 1. Bilateral laryngeal paralysis. *A*, mirror view; *B*, lateral view; *C*, tomogram without phonation; *D*, tomogram with phonation. *i*, ventricular cavity greatly distended. Note: Complete fixation of both cords in adduction.

a tomographic study of a boy, aged twelve, experiencing "change of voice." Both ventricular cavities, true and false cords are well shown here. Failure to visualize both ventricular cavities does not always mean a pathological condition but may be due to the position of the patient such that the axis of the true cord is not perpendicular to the table top. In a normal study both sides should be perfectly symmetrical. If the ventricular cavity is visualized on one side and not on the other, it is an indication that a pathological condition exists. Figure 3, *C* and *D*, is a young woman, aged twenty-six. The inferior cornua of the thyroid cartilage and only a small portion of the body are calcified. The thyroid cartilage calcifies much later in the female than in the male and is often never complete. The ventricular cavity is well shown in the lateral projection. The tomographic study is made posteriorly through the

arytenoids, which are extremely well calcified. This is a very uncommon finding. Figure 3, *E*, is a tomogram of the larynx in a girl, aged eighteen, made during phonation, while *F* is of the same patient without phonation. The cricoid cartilages are well shown here. Normally there should be about 1 mm. of tissue between the cricoid cartilage and the mucosa of the subglottis. This is important as a basis for evaluating subglottic infiltration in cancer of this region. It is noted that the cords lie in quite close approximation during phonation and the ventricular cavities are well filled, whereas when the patient does not phonate the cords are separated and the ventricular cavities seem to collapse.

REPORT OF CASES

Recurrent laryngeal paralysis

Case 1. W. E., male, white, aged thirty-one. Complains of intermittent dyspnea, inspiratory type. Thyroidectomy five years ago, following which patient spoke with a high pitched voice for six weeks. Voice gradually improved and is now almost normal but he has intermittent attacks of inspiratory dyspnea and phonates an almost indescribable low-pitched sound when excited or when he sleeps.

Findings. Mirror view (Fig. 4, *A*) reveals both cords to be fixed in almost complete adduction. The arytenoid cartilages move slightly with phonation. In the lateral roentgenogram (Fig. 4, *B*) there is noted a marked distention of the ventricular cavity. Tomograms (Fig. 4, *C* and *D*), made with and without phonation, reveal the true cords to be fixed. The ventricular cavities are markedly distended when the patient does not phonate but with phonation the false cords are seen to approximate each other and the ventricular cavities partially close. This patient undoubtedly had injury to both the recurrent laryngeal nerves at the time of operation and by some fortuitous circumstance did not develop a complete closure of the glottis and has been able to develop speech under this handicap.

Laryngocele

Kerr and Bradley[2] reported a case of laryngocele together with an excellent résumé of the subject in 1937.

These anomalies are peculiar atavistic structures similar to those occurring in the great apes. They are true herniations of the ventricular cavities and develop from the appendix of the ventricle. In the newborn the appendix is larger than the ventricle itself. It gradually becomes relatively smaller until about the ninth year, at which time the ventricle becomes well developed with a rudimentary appendix.

Case 11. J. M., male, white, aged thirty-eight. Complains of hoarseness. He has had gradual deepening of the voice for one year.

Findings. Mirror examination (Fig. 5, *A*) revealed the left side of the larynx to be normal. On the right side the true cord was not visible. The false cord on the right side and the arytenoid were replaced by a large, non-ulcerated tumefaction which seemed to bulge upward and almost completely obscure the pyriform sinus. The right side was partially fixed. In the lateral projection (Fig. 5, *B*) there is noted a sharply demarcated oval shadow of decreased density which is identified in the tomogram (Fig. 5, *C*) as an air-containing sac on the right side which communicates with the ventricular cavity and produces a marked narrowing of the pyriform sinus laterally and an upward and medial displacement of the false cord and aryepiglottic fold. We note also that there is a rather large compensatory dilatation of the pyriform sinus on the opposite side.

Case 111. E. S., male, white, aged seventy. His chief complaints were hoarseness and dysphagia. The hoarseness was of four and a half months' duration, not severe but becoming progressively worse. The dysphagia was of six weeks' duration.

Findings. Mirror examination (Fig. 6, *A*) revealed a large tumefaction of the right hemilarynx which was non-ulcerated and obscured the true cord on that side. This mass arose under the false cord and aryepiglottic fold producing decreased mobility of that side. The right pyriform sinus was almost completely occluded, while the left was markedly distended (compensatory). A sagittal tomogram (Fig. 6, *B*) reveals a large spherical tumor which seems to be solid and reaches from the hyoid bone to the lower border of the thyroid cartilage. In the coronal tomogram (Fig. 6, *C*) the tumor is seen on the right side, producing marked asymmetry of the supraglottis, and partially obliterating

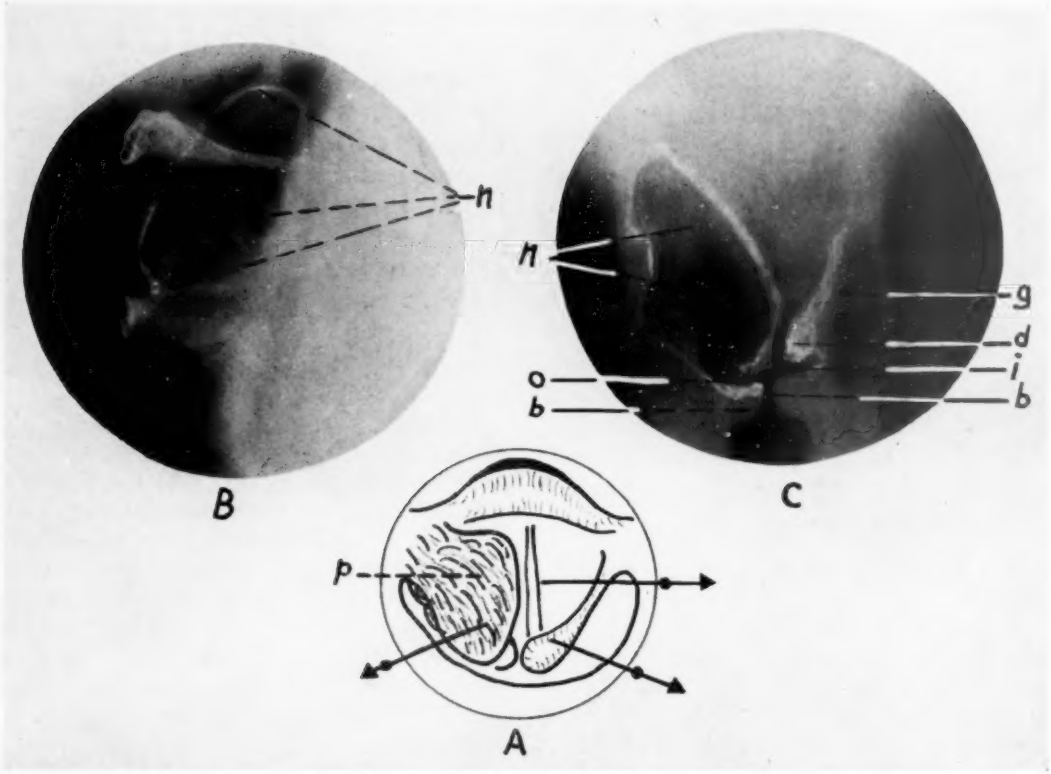

Fig. 5. Case II. Laryngocele. *A*, mirror view; *B*, lateral view; *C*, tomogram. *b*, true cord; *d*, arytenoid cartilage; *g*, pyriform sinus (note compensatory enlargement); *i*, ventricular cavity; *n*, negative shadow of laryngocele sac; *o*, communication with glottis; *p*, tumefaction of right hemilarynx produced by the laryngocele. (Courtesy of Chicago Tumor Institute, Chicago.)

the pyriform sinus. It is seen to consist of two portions: a solid part (*o*) and a less dense part (*n*) which is a continuation of the ventricular cavity and communicates with the glottis.

I have observed this very interesting case for a period of one year, during which time the tumefaction has undergone periodic involution. I assume that the mucus-secreting glands of the ventricular cavity exist in the laryngocele sac and due to obstruction at the outlet are not permitted to escape. This collection of mucus within the sac gives it a solid appearance in the roentgenogram.

Laryngeal Neoplasms

Case IV. W. P., male, white, aged fifty-six. The chief complaints were hoarseness, dysphagia, dyspnea and lump in the neck. The hoarseness was of eight months' duration which was intermittent at first but has recently become constant and progressively worse. Dysphagia was of two months' duration. Gland in the left neck was of seven months' duration.

Findings. Examination disclosed a gland 5 cm. in diameter in the anterior left triangle. Upon mirror examination there was a large mass on the left side protruding upward in the laryngeal vestibule. The true glottis could not be seen. Biopsy report: papillary carcinoma Grade 3. In the lateral view (Fig. 7, *A*) it is noted that the laryngeal face of the epiglottis is entirely smooth. The arytenoid cartilage is quite large and the laryngeal vestibule is partially occluded. In the tomographic study (Fig. 7, *B*) the pyriform sinus on the right side is large and well visualized. The false cord, ventricular cavity, true cord and aryepiglottic fold on this same side appear to be normal, as does also the subglottis. On the left side, the pyriform sinus and the ventricular cavity are entirely obliterated, associated with an asymmetrical deformity of the subglottis. All of this is the result of a tumor mass invading through this area which has, in addition, forced the left

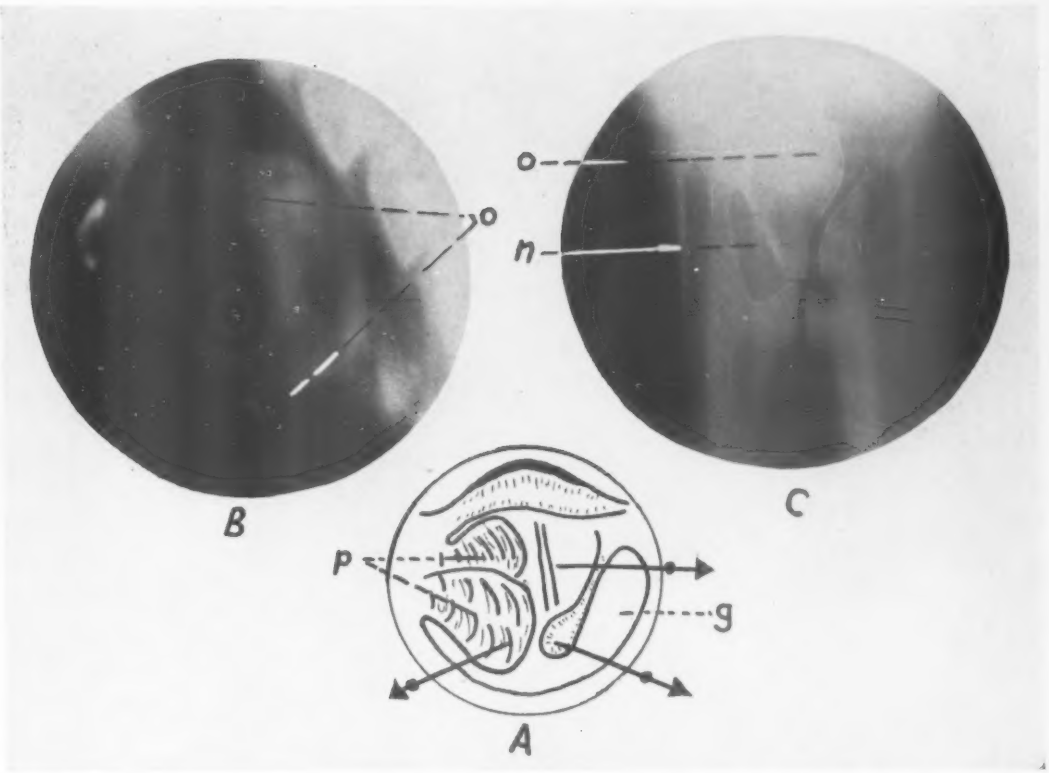

Fig. 6. Case III. Laryngocele. *A*, mirror view; *B*, sagittal tomogram; *C*, coronal tomogram. *g*, pyriform sinus (note compensatory enlargement); *n*, air containing portion of laryngocele communicating with the glottis; *o*, solid portion of the laryngocele; *p*, tumefaction of the left hemilarynx produced by laryngocele.

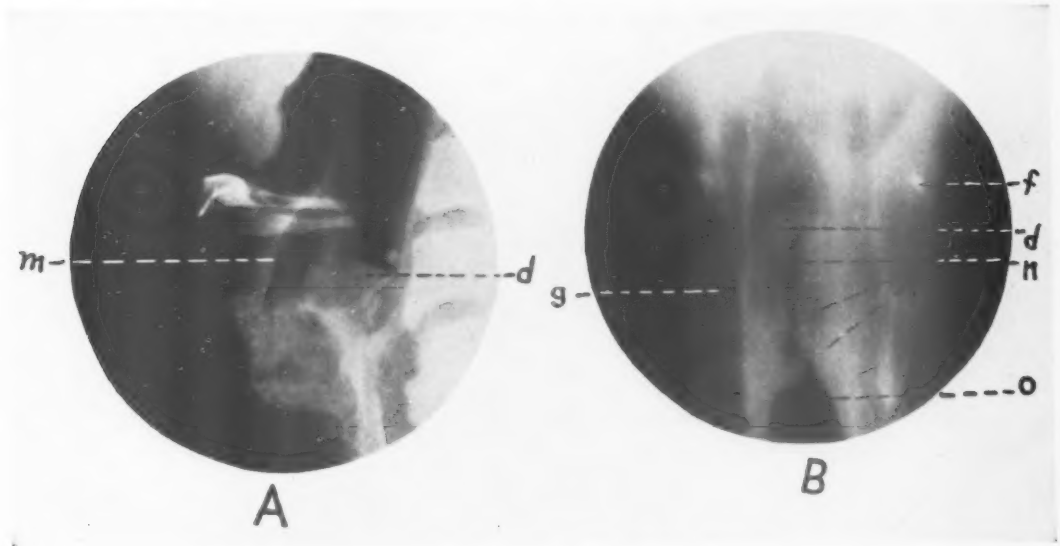

Fig. 7. Case IV. Carcinoma of the larynx. *A*, lateral view; *B*, tomogram. *d*, arytenoid cartilage; *f*, hyoid bone; *g*, pyriform sinus; *m*, laryngeal face of epiglottis; *n*, tumor; *o*, subglottic deformity produced by the tumor.

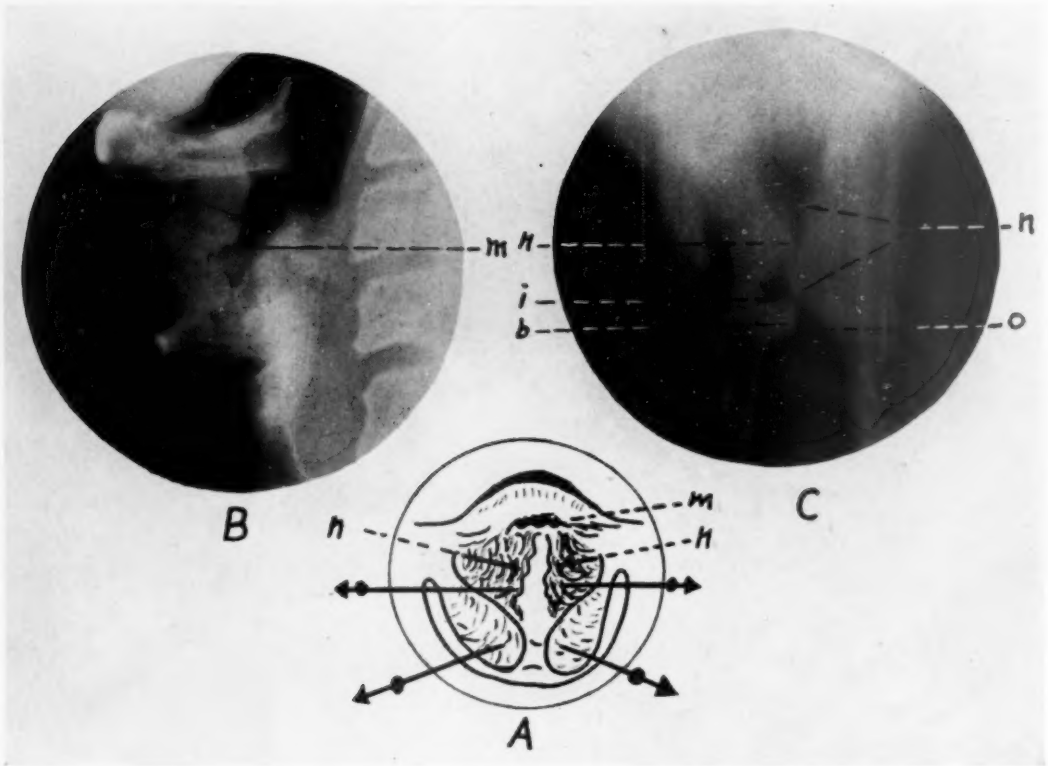

Fig. 8. Case v. Carcinoma of the larynx. *A*, mirror view; *B*, lateral view; *C*, coronal tomogram. *b*, true cord; *i*, ventricular cavity; *m*, laryngeal face of the epiglottis ulcerated; *n*, tumor; *o*, subglottic deformity produced by tumor.

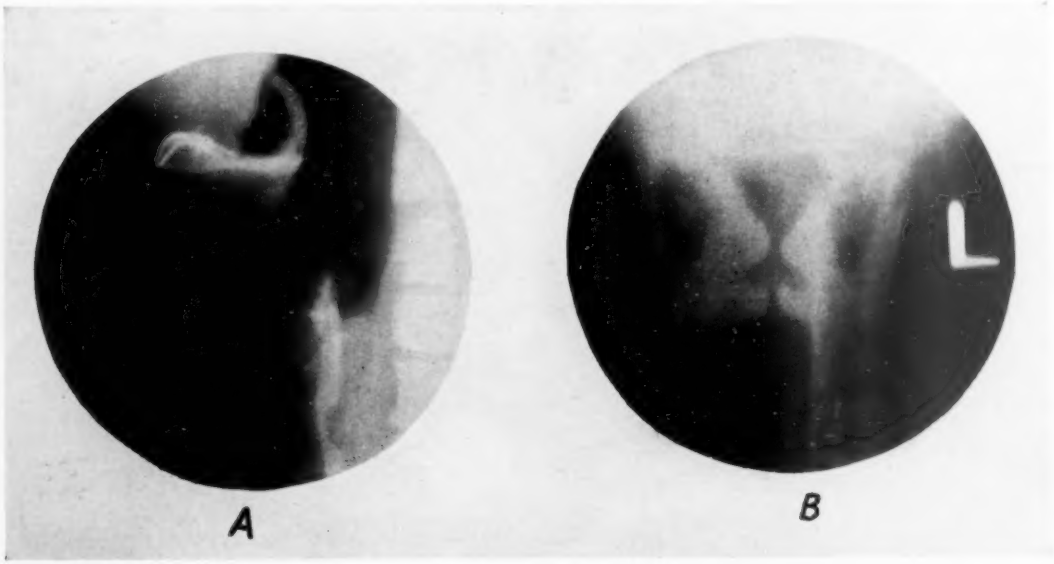

Fig. 9. Case vi. Carcinoma of the larynx. *A*, lateral view (note destruction of the laryngeal face of the epiglottis extending into the pre-epiglottic space. Ventricular cavity is well visualized). *B*, coronal tomogram. Normal except for slight edema of the false cords.

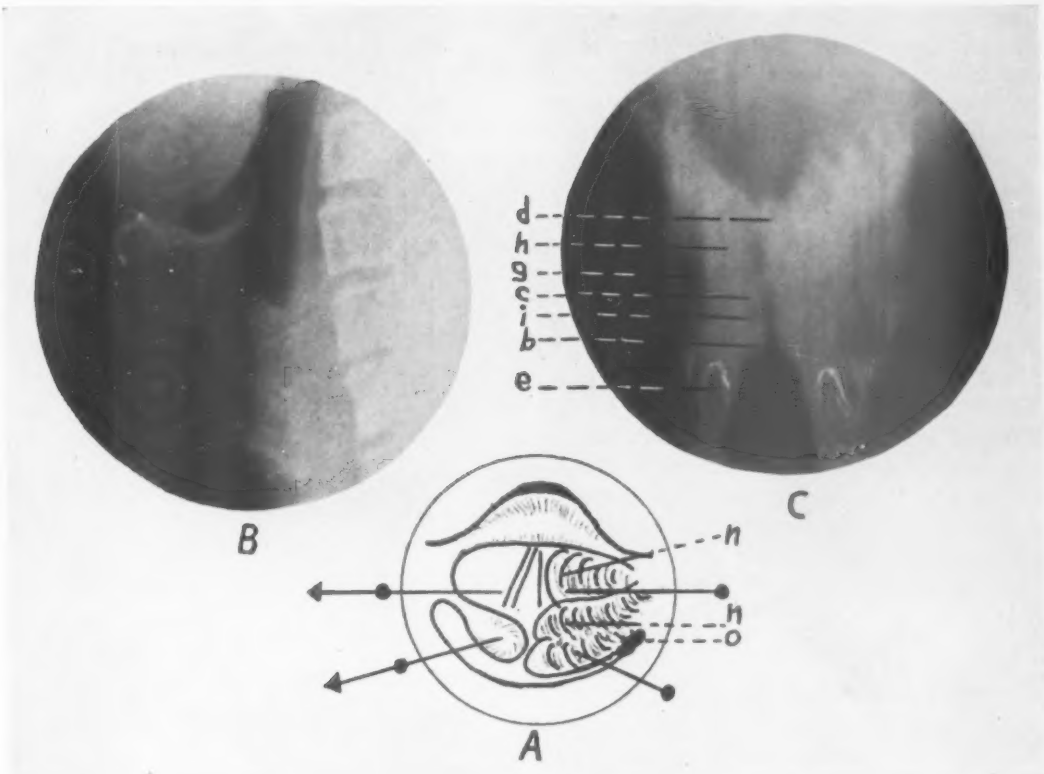

Fig. 10. Case VII. Carcinoma of the hypopharynx. *A*, mirror view; *B*, lateral view; *C*, coronal tomogram (note extensive tumefaction of left hemilarynx). *b*, true cord; *c*, false cord; *d*, arytenoid cartilage; *e*, cricoid cartilage; *g*, pyriform sinus; *h*, aryepiglottic fold; *i*, ventricular cavity; *n*, tumor; *o*, ulceration in pyriform sinus.

arytenoid and aryepiglottic fold upward and medially.

Clinically this is a carcinoma arising in the left ventricular cavity extending downward into the true cord and subglottis, laterally into the pyriform sinus and upward into the false cord and aryepiglottic fold.

Case V. W. K., male, white, aged fifty-eight. His complaints were hoarseness of seven months' duration, intermittent at first but constant during past four months, and dysphagia for the past four months. Left otalgia for one month. Loss of weight of 25 pounds.

Findings. Mirror examination (Fig. 8, *A*) revealed diminished mobility of the entire larynx. Upon the laryngeal face of the epiglottis at the junction with the false cord there was an area of ulceration. The true cords could not be seen due to the tumefaction of both false cords, more marked on the left side. Both arytenoids were edematous, producing considerable narrowing of the pyriform sinuses. Biopsy taken when symptoms were first noticed was negative. In the lateral view (Fig. 8, *B*) there is noted a marked irregularity of the laryngeal face of the epiglottis. The ventricular cavity cannot be identified and there is an opacity of the entire laryngeal vestibule. Tomographic study (Fig. 8, *C*) shows the pyriform sinus on the right side to be partially occluded. The subglottis, true cord and ventricular cavity on the right side are well visualized and normal in appearance. There is a moderate tumefaction of the false cord. On the left side we note that the subglottis is deformed. The true cord and ventricular cavity cannot be identified and the pyriform sinus on this side is completely occluded. The aryepiglottic fold on this side is forced medially and upward.

Clinically this was a carcinoma, probably arising at the junction of the false cord and the laryngeal face of the epiglottis which grew upward along the laryngeal face of the epiglottis, laterally under both false cords, more marked on the left side where it filled the entire ven-

tricular cavity and compressed the pyriform sinus and invaded the floor of the ventricular cavity into the true cord. The biopsy, taken when the patient first had symptoms, was undoubtedly made from the surface of the false cord on the left side and was negative. This is a common occurrence in tumors developing within the ventricular cavities.

CASE VI. C. M., male, white, aged sixty-six. His chief complaint was throat trouble which had its onset six weeks ago with pain low in the throat and upon swallowing. There was intermittent hoarseness of several weeks' duration.

Findings. Upon mirror examination the epiglottis was retracted backward with ulceration on the laryngeal face and edema of both false cords. There was no diminution of the mobility of either side of the larynx. Biopsy showed papillary carcinoma. Lateral view (Fig. 9, *A*) reveals marked deformity and ulceration of the laryngeal face of the epiglottis. The ventricular cavity is clearly visualized. In the tomographic study (Fig. 9, *B*) there is seen no deformity of any of the laryngeal structures except for a very slight edema of the false cords.

Clinically this case is a primary carcinoma originating on the laryngeal surface of the epiglottis destroying the epiglottic cartilage and growing into the pre-epiglottic space. It is interesting to note that in coronal tomography only those lesions which produce deformity in the coronal plane, such as Cases IV and V, are demonstrated, whereas lesions developing anteriorly, such as this last case (Case VI) on the laryngeal face of the epiglottis, or lesions in the anterior commissure and subglottic region are best visualized in the lateral projection.

Hypopharyngeal Lesions (Extrinsic Larynx)

CASE VII. R. H., male, white, aged forty-seven. His chief complaints were sore throat, dysphagia, hoarseness and left otalgia. The pain in the throat had been present for three and a half months, the dysphagia for two months, the hoarseness for one month, and the left otalgia for three weeks.

Findings. Upon mirror examination (Fig. 10, *A*) the right hemilarynx was normal and freely movable. The left hemilarynx was entirely fixed. There was a large tumor involving the left arytenoid, the aryepiglottic fold, false cord, and almost completely obliterating the pyriform sinus which was ulcerated. Biopsy showed epidermoid carcinoma. The lateral projection (Fig. 10, *B*) gives very little positive information. The tomogram (Fig. 10, *C*) reveals the right hemilarynx to be entirely normal. On the left side the subglottis is seen to be entirely normal but the ventricular cavity is obliterated and there is marked upward and medial bulging of the false cord and aryepiglottic fold with obliteration of the pyriform sinus.

Clinically this is a primary carcinoma arising in the left pyriform sinus which had extended medially into the aryepiglottic fold, the false cord and the ventricular cavity.

SUMMARY

Satisfactory tomographic studies of the larynx can be made with simple, inexpensive apparatus giving information that in the past it has been impossible to obtain. It is readily seen that possibilities in the study of the normal larynx present themselves, with a visual concept of alterations produced by disturbances of the nerve supply. Two cases of laryngocele have been shown. It is very doubtful if these interesting lesions have been sufficiently recognized as entities in the past due to the difficulties in diagnosis. Tomographic investigation of laryngeal tumors, when correlated with the history, physical examination and routine roentgen studies, gives further insight into their site of origin and extent of growth.

CONCLUSIONS

1. Tomographic study of the larynx is of more value in lesions producing deformity in the coronal plane than in the sagittal plane.

2. The larynx is placed in the unique position of being one of the few organs in the body that can be readily studied in all three planes, i.e. horizontal, sagittal and coronal.

3. A new source of information is available to the oncologist, the roentgenologist and the laryngologist.*

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* For discussion see page 32.

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TREATMENT OF LARYNGEAL CANCER BY IRRADIATION*

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THREE years ago, in the Janeway Lecture before the Fifth International Congress of Radiology, I reviewed the treatment of laryngeal cancer.[5] Twelve cases were reported in detail to show the trend with radiation therapy over a period of twenty years. The first 6 cases cited were treated with radium and, in these, various surgical procedures had been necessary at one stage or another. The last 5 cases were treated with 200 kv. roentgen rays and an effort was made, with each succeeding case, to show improvement in method. The twelfth case represented the best procedure I had to offer at that time. It is of interest that this entire group of case examples is free from evidence of neoplastic disease now—three years later.

The experience of the three intervening years, in dealing with new material and in observing the cases previously treated, endorses the conclusions drawn in the former publication. The additional time for experience and observation produces certain facts and makes certain impressions which may be of value as a guide in the future.

Any discussion of laryngeal cancer therapy should be confined strictly to the laryngeal box—or what is commonly referred to as *intrinsic* larynx (Fig. 1). That portion of the anatomy of the throat usually referred to as *extrinsic* larynx actually belongs, with one point of exception, to the hypopharynx. The exception is the postcricoid area and this should be classed with the esophagus. Growths, beginning in tissues outside the larynx proper, the intrinsic larynx, are rather generally accepted as being non-surgical. It is important that this distinction, and accurate application of anatomic terms, be adhered to in the present discussion.

In cancer of the true larynx we are dealing with a disease which gives better than average results, surgically or radiotherapeutically, when the best by either method is given to the care of the individual case.

Surgically, and apart from its immediate risk and distress of a very major operation, the permanent disability is obvious, in spite of the various aids to speech. Radiotherapeutically, the indifferent results in recent years which have deterred or discouraged many larnygologists and roentgen theraists have been due to poor technical procedure, fear of adequate dosage, and often the inability to observe accurately and in detail just what is going on in the larynx under treatment day by day.

If there is any value in this brief presentation, it will be by pointing out some of the factors which clinical experience to date has shown to be seemingly essential—even though they may not all be explainable by physical or biological facts now at our disposal.

In the previous publication on this subject, I pointed out that roentgen radiation was the agent of choice in the previously untreated, and preferably early, case. Radium is not the agent for the early favorable case, but may be of vital importance, coupled with laryngofissure, in handling the very advanced case or complications after roentgen irradiation.

In radiation therapy we are rather prone to scan the histopathology of a tumor for some indication of its relative radiosensitivity or radioresistance. This is of value in judging whether we may expect complete or partial regression from a given type of external irradiation. It may be of value in determining whether external plus interstitial or intracavitary irradiation will bring

* Read at the Forty-First Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1-4, 1940.

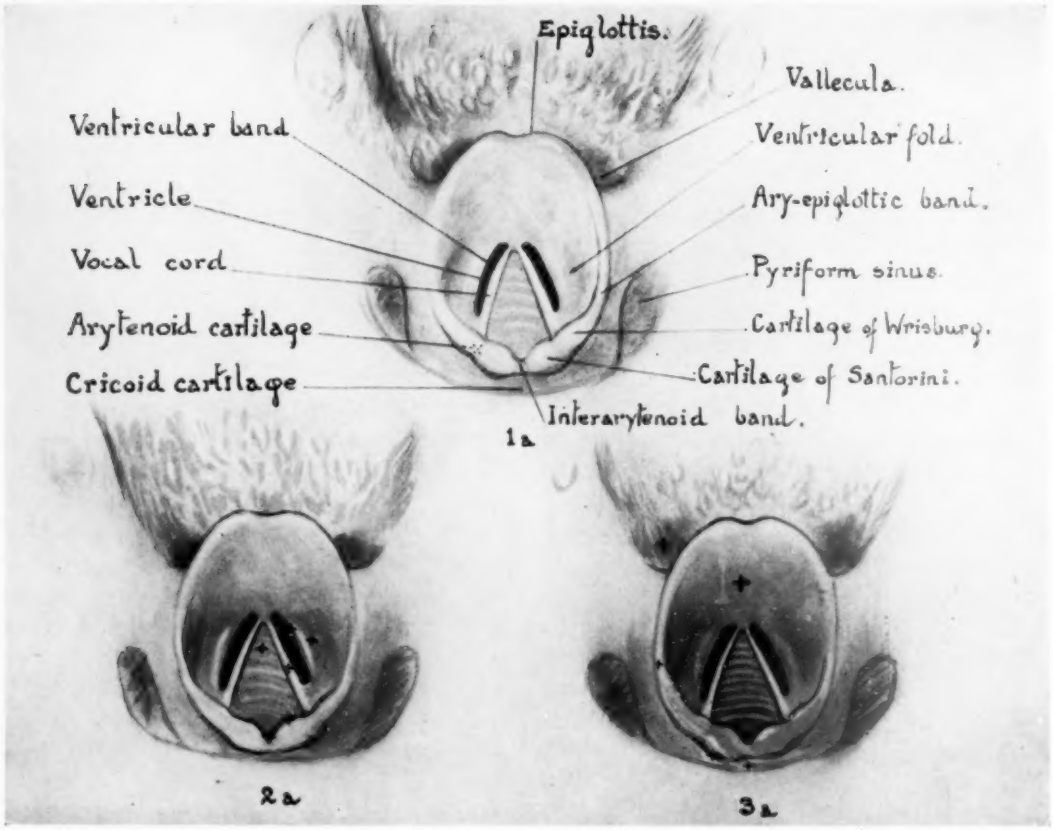

FIG. 1. *1a* shows the anatomic landmarks of importance; *2a* shows by location of marks (*x*) those parts generally accepted as belonging to the larynx proper, usually referred to as intrinsic larynx; *3a* shows the parts usually referred to as making up the extrinsic laryngeal area.

about complete regression. However, where external irradiation only is being used, the usual safe minimum dose is the maximum that the normal structures surrounding will tolerate. This is usually true of laryngeal cancer. The range, however, of laryngeal structure tolerance is considerable in different cases. Hence the necessity for daily examination of the case under treatment. So far, I have not been impressed by any particular features or characteristics of laryngeal cancer histopathology which can be relied upon as a guide to lower or maximum total dosage. As a matter of fact, the histopathologic variation in true laryngeal cancer is more limited than in most anatomic zones.

It is my impression that factors other than histopathology have much more influence over the response of a given laryngeal tumor process to irradiation.

Apart from technical errors, infection plays the most unfavorable rôle. This may be directly obvious or it may exert its unfavorable influence under what might appear to be an independent factor. The laryngeal box represents the narrow point of the respiratory apparatus. In all mammals, except man, its chief function is that of protecting valve to the bronchial tree. In the advanced fungating laryngeal tumor, infection is grossly obvious and the relative response to irradiation is discounted, in some measure, just as is the case elsewhere. In the earlier and more localized growth the effect is not so obvious. However, the anatomic components of the larynx are small and complicated in their

relationships. Infection easily gains access to depth through the ulcerating surface and, once there, is easily harbored. Drainage through the ulcerating surface is incomplete. The lymphatic apparatus is limited, a factor unfavorable to drainage of infection as well as to metastatic extension of the disease. Depth of tumor infiltration is accepted as rendering the irradiation problem more difficult, yet we see surprising regressions at times in very deeply infiltrating tumors at an early stage in the irradiation cycle. We forget that depth of infiltration makes more readily possible depth of permeation of infection.

Damage to cartilage is held up as one of the great hazards of irradiation in cancer of the larynx. The general surgical experience is that cartilage is a very resistant tissue until exposed to infection; it is then, by virtue of its structure, extremely vulnerable. My experience with irradiation in laryngeal cancer has been that the cartilage, as long as it is protected completely by the soft parts, tolerates the irradiation as well as, or better than, the vital normal soft parts. My trouble with cartilage has been where infection has gained access directly to it through the deeply infiltrating tumor and has had no means of adequate drainage. It is under circumstances such as these that radium in conjunction with limited surgery is frequently able to save the situation. It is quite possible that the tremendous advances being made recently with specifics for various infections may be of substantial practical value in conjunction with irradiation in the larynx, and elsewhere.

If the cartilage were directly impaired by irradiation, and out of relative proportion to the soft parts, it is fair to expect much more trouble in young subjects than in elderly patients. With advancing age the hyaline cartilage becomes calcified, leaving only the yellow elastic cartilage free from such change. I have noted no lessening in the supposed cartilage damage in the older group of cases.

The anatomic structure of the larynx, its limited lymphatic drainage apparatus, and complicated attachments of many small independent parts, interferes with drainage, not only of infection, but of tissue exudates independent of infection. This has an influence undoubtedly on the reaction to initial doses of radiation, to total daily doses, to the rate even of daily dose delivery

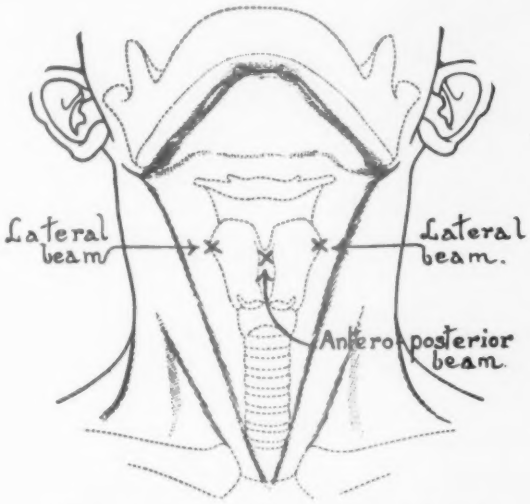

Fig. 2. Diagram showing direction and location of central beams of radiation.

and to the manner of distributing the daily dose. Once the reserve threshold of normal tissue exudate drainage has been overburdened, complicating gross edema results and the ultimate total dose possibility is diminished.

This brings us to a consideration of the technical factors which experience has shown to give the best practical clinical results. This experience has been with 200 kv. and does not extend to higher voltages. In discussing these factors, let us assume an uncomplicated case, viz., one which has not been treated by irradiation or other means and which is not so obstructed as to be in imminent danger of tracheotomy.

Size of Field. I am decidedly in favor of the small rather than large field. The large field covering a good deal of the neck is permissible only in the extremely advanced case which has extended well beyond the larynx proper, with basic laryngeal struc-

ture already gone, and probably tracheotomy. It is an empirical procedure and in the average case subjects far too much of normal neck structure to unnecessary irradiation. Some of my material has been treated using a 3 cm. circular field and some with a 4×5 cm. rectangular field. The latter impresses me more favorably although I am not committed to the exact size. I do favor the square or rectangular field rather than the circular, and I can see practical physical reasons for using a narrower anterior field. Two lateral fields and one anterior field have, in my experience, permitted of the most uniform irradia- of the laryngeal box (Fig. 2). The total dose, maximum, found to be effective can be delivered without more than transient skin damage. The skin and subcutaneous normal tissue change can be safeguarded still further by slight angulation of the lateral beams forward or backward, depending on location of growth. I have not found posterior fields necessary or of value. In adjusting small fields it is essential that it be done by one familiar with the anatomy in general and the individual case in particular and then only after a mirror examination of the patient on that particular day. It is not fair to the patient to leave such adjustment to a technician.

Distance. Contrary to physical facts with which we are all familiar, I still adhere, on the basis of clinical observations, to a distance of 80 cm.

Filter. At 200 kv. I am convinced the minimum filter for laryngeal work is the Thoraeus filter or its equivalent in copper, plus whatever other essential filter the particular equipment used interposes. My best results have been with a filtration equivalent to 2.32 mm. Cu.

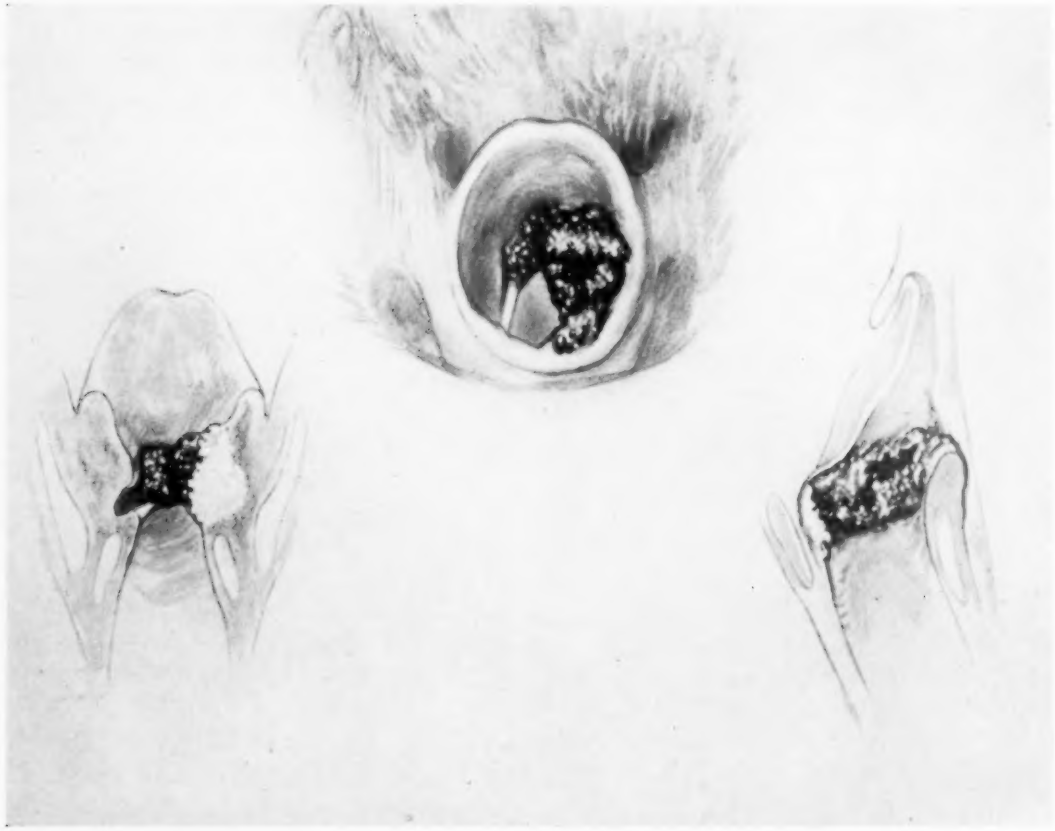

Fig. 3*a*. Visualization of location and extent of growth.

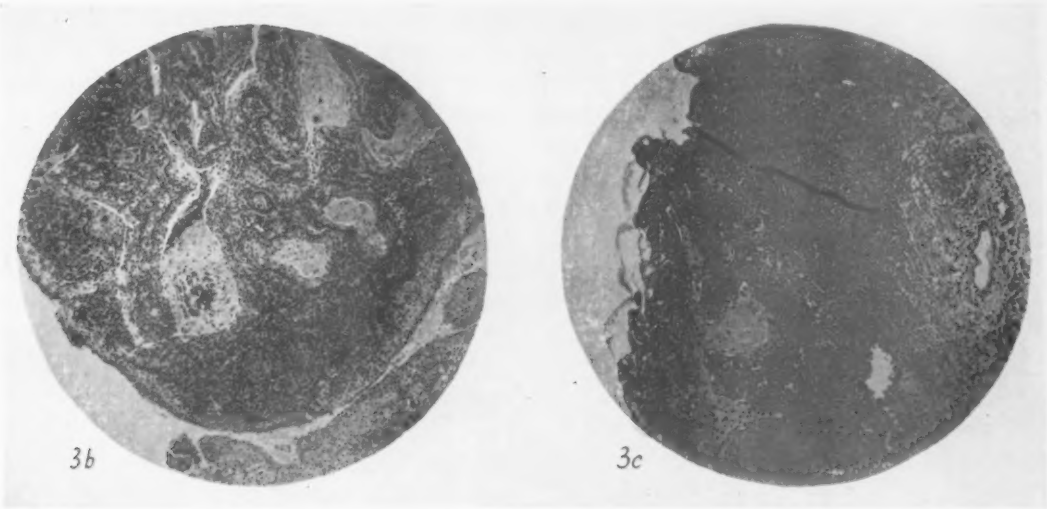

Fig. 3*b*. Photomicrograph showing histopathology of growth before treatment.
Fig. 3*c*. Photomicrograph showing histopathology of tumor-bearing area after treatment with large dosage; tumor remnants still to be found.

Roentgens per Minute. This is a matter of substantial economic importance—both time and cost. Distance and filter impose, with the average 200 kv. equipment, a relatively low dosage of roentgens per minute. If one or the other or both of these factors are lessened, the relative reaction, clinically, is still more favorable with a low dosage of roentgens per minute as against the maximum which the equipment will deliver. I have continued, by trying various combinations of distance, filter, and roentgens per minute, to bring the clinical reactions into line with the physical facts as given us by the physicists. So far I have not been able to duplicate our best reactions by such combinations as maintaining a low dosage of roentgens per minute, 80 cm. distance and 1 mm. Cu, or less, of filter or 50 cm. distance with 2 mm. Cu or more of filter. The dosage in roentgens per minute which we have found to be most acceptable has been 6 to 7 r and the optimum may be well below this level. It is interesting to speculate on the influence of this factor, as well as those yet to be mentioned, on the rate of development of cellular and intercellular edema in the tumor-bearing area.

Daily Dose. We have found it best to begin with a low initial daily dose—50 r to each of the lateral fields, gradually increasing to 75 r to the lateral and anterior fields until during the second week each of the three fields is receiving 100 r. This treatment is on the basis of five days a week. So far, I have felt that the benefit to the patient's general economy, by reason of the week-end rest, more than outweighed the theoretical effect of interrupting the daily treatment. Exposure of the three fields each day gives a more uniform distribution and clinical reaction than treating the fields in daily sequence. The daily dose of 300 r, with the factors mentioned, is the maximum that we have found acceptable, even though it would seem that much more might be tolerated on any given day.

In my previous publication detailed case reports were given, in sequence, to show the results and benefits from varying and improving these various factors.

Total Dose. This is a decidedly variable quantity and must be judged from observation of the individual case. Treatment should not be stopped when symptoms are relieved and the surface appears epithelialized at a low total dose level. In my original

report,[5] Case VIII showed a peculiar result with very low total dose and inferior secondary factors. He is still well nearly ten years later. However, we have no duplicate of such a reaction. It would seem that, with the other factors and spacing I have used, 7,500 r total is about the minimum safe dosage. We have not had difficulty, with the same factors and spacing and in more slowly reacting tumors, in giving as much as 12,000 r total over a sixty day period. We have gone beyond that and have gotten into trouble, as in a case which I shall later cite.

Total Time. With the data given, it is evident that the minimum total dose advised requires about forty days, and the maximum about sixty days for delivery. This is scarcely in keeping with the optimum reaction period of epidermoid carcinoma as reported by various observers concerned with optimum reaction without reference to location. The larynx will tolerate just so much per day—beyond that lies trouble.

Supporting Measures. A patient subjected to heavy prolonged irradiation requires every possible supportive measure, both locally and generally. Frequent irrigations and sprays with aqueous and oil solutions keep the throat clean and relatively comfortable. Cleansing and relief of nasal obstruction promotes nasal breathing and results in less drying of the mucosa than is the case in mouth breathing. Vigorous parenteral medication with liver extract and vitamin B complex maintains the blood picture at a level which permits of the most favorable reaction to irradiation. A non-irritating, high-caloric, balanced diet must be maintained.

At this point the question may well be

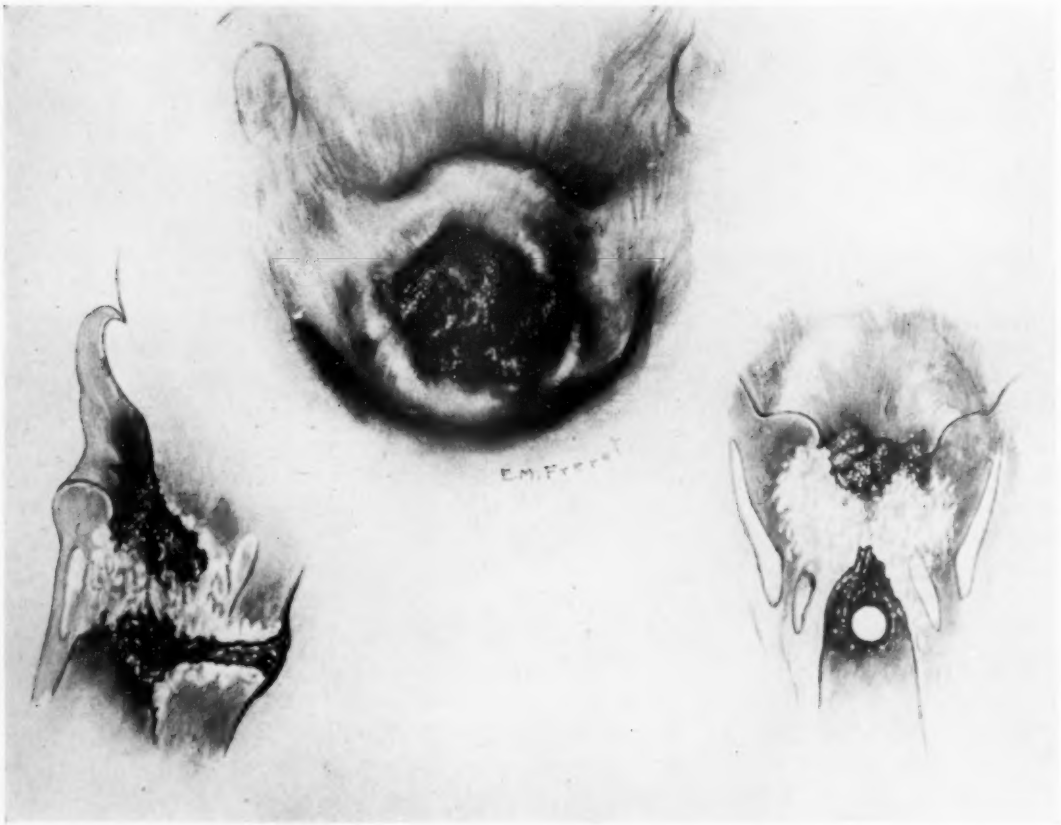

Fig. 4*a*. Type of case suitable for laryngotomy and radium with or without subsequent cautery.

asked—why go into all of these details? The answer is that carcinoma of the larynx, where the basic structure of the larynx is still intact, is curable in a high percentage of cases by roentgen irradiation alone without hospitalization or interim risk and without loss of voice. The reason why that statement is not generally accepted and that result not obtained with reasonable regularity is because of failure to observe these various details and often inability on the part of the radiotherapist to properly examine the patient from day to day while under treatment.

It is obvious that the patient must be cooperative in regular attendance for treatment and attention to his part of the detail. It is not applicable to cases in which other methods, or radiation therapy of a sort, have been tried, or started and discontinued.

If for any reason the surrounding normal structures have been damaged previously —recurrence or incomplete irradiation—or if the case is advanced to the point of imminent obstruction requiring tracheotomy, it is not one for roentgen therapy alone aimed at complete restoration of function. Roentgen irradiation may be used as an initial step but usually preliminary to thyrotomy or laryngofissure for the purpose of direct implantation of radon. A good many advanced or indifferent therapy complicated cases can be salvaged by this means, but they usually have permanent tracheotomy and loss of voice. They are outside the scope of radical operation and hence represent total salvage but the result is the same as though accomplished by a total laryngectomy.

Case v in my original report[5] is an example of ill-advised roentgen therapy followed by ultimate cure by tracheotomy, laryngofissure, and cleaning up of debris with the bipolar cutting cautery. He was treated by radon implants in 1926—complete regression, no secondary necrosis and perfect restoration of function. In 1933 a recurrence at the border of the old scar was treated by roentgen irradiation. This was

Fig. 4*b*. Histopathology of tumor. Patient now well thirteen years.

an error because of the previous irradiation. The immediate results appeared to be highly satisfactory but seven months later, following a severe cold, an acute inflammatory reaction necessitated an emergency tracheotomy. Two days later a laryngofissure was done and the infected, partially broken down soft parts and cartilage removed. The patient remains well and uses an artificial speaking apparatus to excellent advantage. However, the use of intensive roentgen irradiation after his initial radium treatment was an error. The recurrence could have been handled by thyrotomy and radon implants much more safely with less discomfort and a much shorter period of disability. The ultimate tissue damage and tissue loss would have been less.

Case iv of the original report is a good example of the use of tracheotomy, thyrotomy, cautery and radon implants in an advanced case beyond the scope of total laryngectomy. She is now well over thirteen years (Fig. 4*a*).

Very occasionally one finds an extremely radioresistant case, usually accompanied by a stubborn low grade infection in the tumor-bearing area, which has not re-

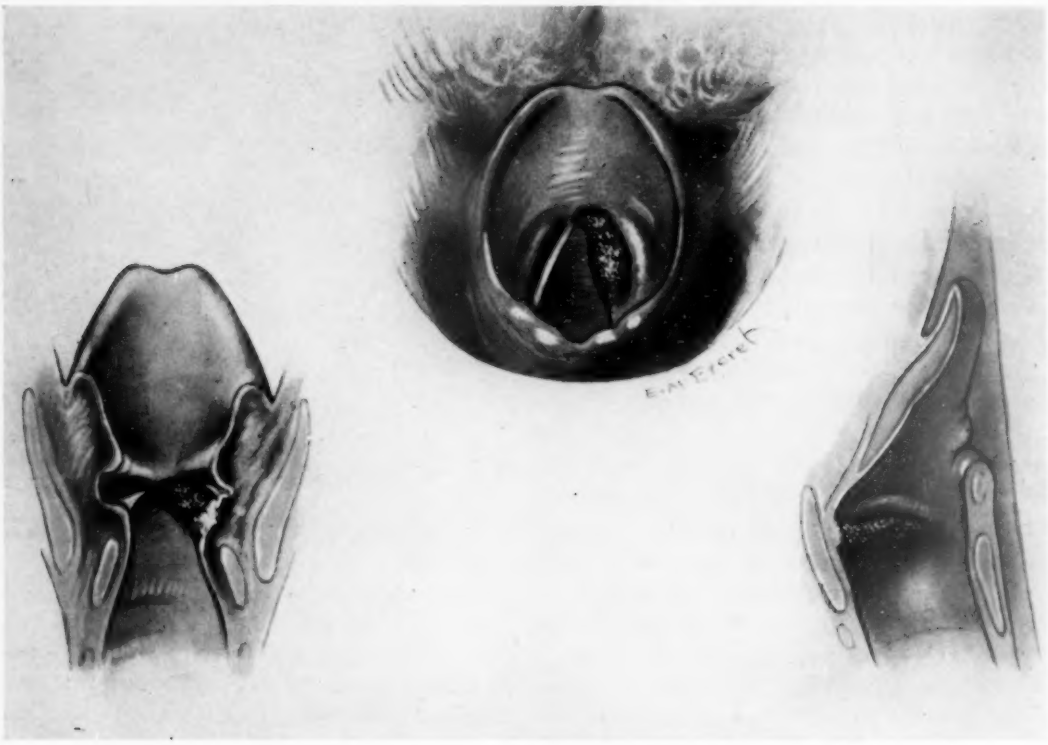

Fig. 5*a*. Visualization of extent of growth.

gressed completely after a total dosage of 12,000 r. I believe it is best to refrain from further roentgen irradiation and resort to thyrotomy and radon implants or the cutting cautery, depending upon local findings at operation. The case is a failure for roentgen irradiation but not necessarily a loss if handled promptly, and the ultimate result is the same as with laryngectomy. Figure 3*a* is an example of such a case. A male, aged thirty-seven, with Grade 2 squamous cell carcinoma involving the entire right cord, right arytenoid and one-half of the left cord had received a total of 12,000 r over a seventy-four day period. The total period had been lengthened by reason of a troublesome inflammatory reaction from local infection. Tumor tissue was still present and the error was made of continuing the roentgen irradiation—the skin was in excellent condition. The dose was increased to a total of 18,750 r. Six weeks later hoarseness increased and after another two weeks tracheotomy was necessary. Laryngofissure was done and the entire area cleaned out. Healing was slow but complete and he mastered the use of an artificial speaking apparatus very well. After two years he developed a recurrence around the tube and died. He was in another country, so that we were not able to get satisfactory details of the recurrence or its treatment. It would have been much better to have stopped the roentgen irradiation at 12,000 r and resorted to thyrotomy plus radon implants or cautery, leaving the patient with a permanent tracheotomy.

In the group of case examples cited three years ago, only one has since developed a metastatic node. Case XII, a left cord growth, developed a palpable node just above and back of the greater cornu of the hyoid bone, left side, in the spring of 1939. It was cared for by our usual procedure of surgical exposure and radon implantation and has given no further trouble. In the

fall of 1939 he returned with an independent primary growth on the posterior surface of the epiglottis near its tip. This was treated with radium. Both the metastatic node and the second primary growth were just outside the field of the original irradiation. This might be used as argument in favor of larger fields for laryngeal irradiation. A sporadic case, however, is not enough to overweigh the other disadvantages of larger fields. The patient was the heaviest cigarette smoker I have ever met and his entire upper mucous membrane tract showed marked changes. The gold seeds in the metastatic node did not excite a secondary reaction in the previously treated larynx.

It is seldom that we have the opportunity of studying, outside the body, a larynx previously treated successfully by roentgen irradiation. Figure 5*a* is a photograph of the gross specimen of such a case. A male, aged sixty-two, had a Grade 2 squamous cell carcinoma involving the anterior two-thirds of the right cord extending subglottic and into the anterior commissure but not involving the left cord. He was treated by the factors which we have previously outlined as being most favorable thus far in our experience. He received a total of 12,200 r over a period of sixty days. The course under treatment was uncomplicated. Healing was complete with no symptoms of irritability or dryness. About six months later he was admitted to another hospital with an acute obstruction of the bowel which proved at autopsy to be an adenocarcinoma of the sigmoid with adenocarcinoma, metastatic in both liver and lungs. There was no residuum of laryngeal cancer at the primary site or metastasis elsewhere.

Fig. 5*b*. Photomicrograph showing histopathology of the growth.

Fig. 5*c*. Autopsy specimen: six months after treatment of the larynx. Perfect healing—no remaining tumor.

SUMMARY

Carefully done roentgen therapy offers an excellent chance for cure in laryngeal cancer uncomplicated by previous treatment.

This method is applicable to a larger group than is radical surgery.

This method is free from interim risk

and results in complete restoration of function.

There is a limit beyond which it is unsafe to increase the roentgen-ray dosage.

With these cases resistant to roentgen irradiation, recurrences, and incompletely treated cases, laryngofissure plus radon implants or cautery offers a chance for further cure but without restoration of function in all except an occasional case.

Metastatic cervical nodes are treated by surgical exposure and radon implantation; their occurrence is infrequent.*

* For discussion, see page 32.

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CAUSES OF FAILURE OF ROENTGEN THERAPY IN CANCER OF THE LARYNX*

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FAILURE to cure cancer of the larynx by roentgen therapy may be due to local causes in the region of the tumor, general causes associated with the inability of the patient to tolerate the treatment, and technical causes due to faulty technique. Among the local causes may be mentioned extensiveness of the growth, invasion of cartilage as well as deep local infection and necrosis. In the group of general causes may be placed decompensated cardiac disease, chronic alcoholism and any chronic debilitating disease which reduces the patient's tolerance to support the unavoidable radiation pharyngitis and laryngitis. Among the technical faults should be listed too intensive daily or too great total roentgen-ray dosage and too large fields, or on the contrary, underdosage and inadequate fields.

CLINICAL MATERIAL

In an effort to gain a clearer appreciation of these various causes of failure, a study was undertaken of 166 case histories, that is of all patients treated with roentgen irradiation for cancer of the larynx between 1926 and 1938 in the Departments of Radiotherapy of Presbyterian Hospital and of Montefiore Hospital, New York City. When reviewed in July and August, 1940, the 166 patients were classified as follows: 118 patients dead (D), 13 alive with disease (A.D.) and 35 free from clinical evidence of cancer (S.F.). Among those classed as dead, there were a number who may be free from cancer but who were lost to follow-up and 12 who died of intercurrent disease, though clinically free from cancer. Many patients, classed as treated, did not complete the treatment but were given only a few palliative exposures because of the advanced stage of their disease. No deductions of any kind were made in the statistics.

One hundred and fifty-nine of the patients were males and 7 were females. Sixty-six per cent of the patients were between fifty and seventy years, 16 per cent between forty and fifty, 11 per cent between seventy and eighty, and the few remaining cases were scattered through the other age groups.

Microscopic proof of epidermoid epithelioma was obtained in 157 of the 166 patients. In 8 of the 9 unbiopsied cases, the correctness of the clinical diagnosis was supported by the fact that they subsequently died with symptoms and signs suggesting cancer. The remaining patient represents the only microscopically unproved case now classed as "free from symptoms." Five and one-half years ago he had a small lesion in the middle of one vocal cord and the clinical diagnosis of cancer was made independently by two recognized laryngologists.

One hundred and twenty-three patients were treated with roentgen therapy alone. In 43 other patients, roentgen therapy was given as an addition to other irradiation or to surgery. Analysis of the causes of failure is much more difficult in the latter group because of the complication of the additional treatment. The latter material has therefore been considered only in passing, while the cases treated by a single series of roentgen exposures alone have been analyzed from the following standpoints: microscopic structure, assumed site of origin of cancer, extent of local and regional involvement and result of treatment.

FACTORS INHERENT IN THE REGION OF THE TUMOR

Microscopic Classification. Seventy-eight

* Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

of the 123 cases treated by roentgen therapy alone were recently reclassified microscopically, according to the differentiation of the epitheliomas, by Dr. A. P. Stout, the surgical pathologist of the Presbyterian Hospital. Slides from the unclassified cases came mostly from other hospitals and had been studied, but not classified, at the time of admission of the patient for roentgen therapy. These slides were not available at the time when the grading was done. Many border line cases had to be classified in a middle, large group designated as "medium differentiation" and only a small number of cases were left for the other two groups. A comparison based on these numerically divergent groups was not considered of much value. Harris and Klemperer[4] were among the first to question the bearing of the microscopic structure on radiosensitivity in cancer of the larynx. Coutard, the father of modern roentgen therapy of cancer of the larynx who originally laid great emphasis on the microscopic structure of the treated tumors, paid even greater attention to fixation and especially to invasion of the arytenoid region. Cutler,[3] who has recently reviewed this question, feels that mobility of the tumor is more important than its histology. The bearing which differentiation of epithelioma may have on results of roentgen therapy in cancer of the larynx cannot be deduced from data available in our series and presented in Table I.

Table I

78 cases

	Total	Dead	A.D.*	S.F.†
Well differentiated	14	8	1	5
Medium differentiated	49	39	3	7
Poorly differentiated	15	10	1	4

* Alive with disease.
† Free from clinical evidence of cancer.

Site of Origin. The location of the primary lesion is easily discernible in cases in which the lesion is early and small. In extensive involvement, as emphasized by Coutard, the primary site may be recognized if the growth is observed while it regresses under roentgen therapy, as the primary lesion is usually the last one to clear up. Table II represents classification of 123 patients according to the assumed site of origin. In Quick's[10] excellent Janeway Memorial lecture, the author who limits his discussion to cancers arising "in the larynx proper, usually referred to as intrinsic larynx" intentionally excludes cancer originating on the epiglottis, its folds or on the arytenoid. If one considers separately the characteristics of growths arising in these various locations, there should be no objection to including them in a discussion of cancer of the larynx.

Table II

Results after roentgen therapy only
(123 patients)

Assumed Site of Origin	Total Number of Cases	Dead	A.D.	S.F.
Vocal cord	55	39	3	13
Band	9	7	0	2
Subglottic	3	2	0	1
Epiglottic	25	17	3	5
Aryepiglottic	5	3	0	2
Arytenoid	26	24	2	0
	123	92	8	23

Invasion of Cartilage. From the above figures it will be noted that the results were poorest in cancers involving the arytenoid cartilages. The mucosa is close to cartilage throughout the greater portion of the larynx. Deep ulceration of a cancer on the mucosa may permit the entrance of the ever present bacteria, and necrosis of the cartilage may result. In necrosis of the epiglottis, drainage is downward and the necrotic material is emptied towards the ventricle, whence it is coughed up and expectorated. Cure may result from roentgen therapy even if an epiglottis invaded by cancer should undergo complete necrosis after this treatment. This does not usually hold true for similar necrosis of the arytenoids. The arytenoid cartilages are located in a sort of a pouch to one side of the air

column, which is expelled in coughing. Drainage is upward and coughing does not help the emptying of this deep, infected cavity. This is probably the reason for the poor results recorded after roentgen therapy of cancer of this location, whether the involvement of the arytenoid be from a primary lesion on the superimposed mucosa or is a secondary extension from a primary tumor on the true cord. Kramer and Harris,[8] however, have reported a small series of cases with fixed arytenoids presumably invaded by cancer, in whom the results of roentgen therapy were satisfactory.

If it is assumed that in the majority of cases fixation of a cord is due to invasion by cancer and not to inflammation, the results given in Table III show the relative gravity of this complication in our series. It should be emphasized, however, that among 33 patients with fixed cords 6 are now free from clinical signs of cancer.

Table III

PROGNOSTIC SIGNIFICANCE OF FIXATION OF THE CORD

	Total	Dead	A.D.	S.F.	
Cords fixed	33	24	3	6	(19.9%)
Cords not fixed	19	13	0	6	(31.5%)

Extent of Tumor.

True cord. The local extent of the tumor naturally influences the outcome of the treatment. In epitheliomas of the true cords

Table IV

EXTENT AND PROGNOSIS IN CORDAL EPITHELIOMAS

	Total	Dead	A.D.	S.F.	
Cancer limited to one cord	9	4	0	5	(56%)
Cancer not limited to one cord	46	34	4	8	(19.5%)

limited to one cord, the results are better than in those not so limited, as seen in Table IV.

Of those limited to one cord, one died of cardiac failure, though free from laryngeal cancer; another was well when last seen in the spring of 1938, but was classified as dead as he was then lost to follow-up. The only 2 patients with a single cord involved, known to have died of cancer of the larynx, originated in the posterior one-third of the cord where hidden extension to the arytenoids may have easily occurred. Thus in a small number of cases limited to the anterior two-thirds of one cord, the results of properly applied roentgen therapy have been as good as those of laryngofissure, which is the treatment usually chosen. Coutard[2] and Quick[10] agree with this view, whereas Martin,[9] Imperatori,[5] and others, believe that all intrinsic cancers should be treated surgically. We believe surgery and roentgen therapy are equally efficient in these cases and allow the patient to choose the treatment which he prefers. Roentgen therapy takes five to six weeks, the reactions of the mucous membrane and skin are severe and painful, but the resulting voice is better than after laryngofissure. The wound after laryngofissure heals promptly and the patient is back home in a week, but the resulting voice is husky and low pitched. Theoretically, the curability rate with roentgen therapy should in this variety of laryngeal cancer be higher than after cordectomy. The zone of effective roentgen therapy includes the entire larynx, i.e., also clinically unappreciable extensions beyond the cord, which may escape removal, if excision is limited to the cord. As yet an insufficient number of cases have been treated by roentgen therapy to permit a fair comparison but logically it should be superior to cordectomy.

In the more extensive cases in which laryngectomy is the usual procedure, the results after roentgen therapy are correspondingly worse. In some cases it is difficult to decide whether to choose surgery or roentgen irradiation. In these cases we have recently been trying out a new approach: If there is definite shrinkage of the tumor after half or two-thirds of the full dose has been administered, we continue with roent-

gen therapy, otherwise we advise laryngectomy.

Epiglottic cancer invading tongue. In cancers arising on the anterior surface of the epiglottis, deep extension to the base of the tongue frequently means failure. Cures are more frequent in fungating tumors not invading the tongue, even if they have destroyed the epiglottis and have metastasized to the cervical lymph nodes. The relative gravity of invasion of the tongue is shown in Table v.

Table V

INFLUENCE OF INVASION OF BASE OF TONGUE

	Total	Dead	A.D.	S.F.
Tongue involved	7	6	1	0
Tongue not involved	18	11	2	5

Tracheotomy. The need for tracheotomy in patients with cancer of the larynx is of grave significance. Prognostically, it does not seem to matter whether the tracheotomy is performed before, during or after roentgen therapy. If performed shortly before or soon after starting roentgen therapy, this suggests that the disease is extensive. If done after completion of irradiation, it is performed because the disease has remained extensive or because of post-irradiation edema of the laryngeal structures. The latter occurs chiefly where the daily or total dosages have been too large. Only 2 of the 42 patients in whom a tracheotomy was performed remained well as is shown in Table vi.

Table VI

PROGNOSTIC SIGNIFICANCE OF TRACHEOTOMY (42 PATIENTS)

	Total	Dead	A.D.	S.F.
Before roentgen irradiation	13	12	0	1
During roentgen irradiation	5	5	0	0
After roentgen irradiation	24	23	0	1

Lymphadenopathy. Patients arriving for treatment with palpable cervical lymph nodes have extensive disease and the results in these cases are poorer than in those without nodes. This is illustrated in Table vii.

Table VII

PROGNOSTIC SIGNIFICANCE OF NODES ON ADMISSION (123 PATIENTS)

	Total	Dead	A.D.	S.F.	
Clinically involved	60	47	5	8	(10.3%)
Clinically not involved	55	39	2	14	(24.5%)
Doubtful	8	7	0	1	

The relative frequency of clinical involvement of regional nodes and their prognostic significance differed in the various anatomic varieties, as shown in Table viii. Patients with palpable nodes did not do as well as others in the same group whose nodes were not palpable. There were some of the former who remained well in spite of their more extensive involvement. The diagnosis of metastasis was on clinical grounds and microscopic proof of metastasis was not obtained in these nodes.

Table VIII

PROGNOSTIC SIGNIFICANCE OF LYMPH NODES ON ADMISSION ACCORDING TO SITE OF PRIMARY LESION

	Lymph Nodes	Total	Dead	A.D.	S.F.
Vocal cord	without	33	21	2	10
	with	19	17	1	1
Band	without	3	3		
	with	6	4		2
Subglottic	without	2	2		
	with	1			1
Epiglottis	without	4	2	1	1
	with	20	15	2	3
Aryepiglottic	without	4	3		1
	with	1			1
Arytenoid	without	5	5		
	with	17	15	2	

FAILURES DUE TO THE GENERAL CONDITION OF PATIENT

Debilitated patients suffering from chronic infection, arteriosclerosis, advanced nephritis, cardiac disease, diabetes or chronic alcoholism may not be able to withstand the treatment and especially not the reaction which follows. Dosage in them is therefore reduced and an inadequate amount administered resulting in failure.

FAILURES DUE TO TECHNIQUE

Underdosage. Failures during the early years of roentgen therapy were due mainly to underdosage. Small quantities of roentgen rays were given with low voltage machines, repeated at weekly or monthly intervals. These temporarily controlled the disease but clinical freedom from cancer for five years was either unknown or occurred only exceptionally. The skin and mucosae did not show the severe acute reactions, regarded as normal nowadays. The late reactions, however, were more noticeable than those from modern treatment. Though late sclerosis of the connective tissues, telangiectasia and skin atrophy resulted from cumulation of the weak doses administered at long intervals, the cancer was not cured as at no time was it given a sufficient amount of treatment to destroy it. It was not so much the low voltage as the inadequacy of the total dose within any one period that made the treatment inefficient. It should be mentioned in this connection that the first few cases cured by Coutard and surviving five and ten years were treated with 130 kv. filtered through 3 mm. Al. The dosage, however, was much greater than that referred to in the discussion above.

Overdosage. After this period of inadequate dosage, came the period when an attempt was made to give very large dosage in short exposures; these were occasionally repeated. This method of roentgen therapy, practiced by Jüngling,[6] and others, produced severe, acute irradiation reactions in the normal tissues. It was frequently complicated by edema of the arytenoids and necrosis of the laryngeal cartilages. Rare cures may have occurred but were insignificant in number. In an effort to reduce this injury to the normal laryngeal tissues and yet retain the principle of a large total roentgen-ray dose necessary for the destruction of the cancer, Coutard at the suggestion of Regaud developed his protracted fractionated method. In this method, as in Pfahler's saturation method, a large total dose is subdivided into multiple small daily fractions. Coutard's method differs from Pfahler's in the fact that the total quantity of roentgen rays is larger and that the series of treatment is given only once and not repeated. Instead of the three weeks used for saturation, Coutard now takes six to seven weeks to administer his treatment.

Our own experience with roentgen therapy closely parallels the developments just outlined. While most of the clinical material during the first five years consisted of hopelessly far advanced cases in whom little could be accomplished, many mistakes were also made in the technique of the treatment. Some of these have been remedied during the last few years.

Dehydration. Inadequate dosage accounted for several failures during the early years. In others, in whom the dosage seemed adequate, the dysphagia which follows the irradiation reaction was allowed to lead to dehydration. This may have contributed to the fact that several patients died from aspiration bronchopneumonia. The danger of dehydration has been partially overcome by occasional hospitalization during and immediately after the period of severe laryngeal reaction. This permits control of the body water level by the administration of fluids by gavage, per rectum, subcutaneously or intravenously. Up to 1930 severe reactions of the laryngeal mucosa were thought to be essential for a cure. Since 1931 severe reactions have been avoided and an attempt made to obtain only a thin mild pseudodiphtheritic membrane. In a personal communication Bac-

lesse[1] years ago questioned the advisability of a thick heavy pseudodiphtheritic reaction membrane. Stebbing[12] and Juul and Strandberg[7] felt it was indicated only exceptionally.

Excessive Dosage and Fields. Total doses of 4,500 r in twenty days with lateral fields of 10×15 cm. on each side of the larynx were often used in our clinics up to about 1931. Early edema of the arytenoid region, marked dysphagia and later chondronecrosis were common. In later years the total amounts were reduced to about 3,000 r to 3,200 r/o to each of the two 6×8 cm. lateral laryngeal fields. We have not had any cures where the total dosage has been less than 2,700 r/o to each of the two lateral fields. In more extensive cases 8×10 cm. fields are still employed for the first 500 to 750 r and the fields then reduced to 6×8 cm. so as to embrace more closely the clinically appreciable extent of the cancer. Fields of 10×15 cm. are not used any longer except for regional metastases and dosage is then kept relatively low. In lesions limited to the cords, fields 5 cm. in diameter have occasionally been employed but this has not been the rule. The treatment period has been raised from the original twenty to forty, or more days, and the daily dose has been reduced from 400 r/o to one field to 100 r/o or 125 r/o to each of the two lateral fields. Treatment is discontinued for a few days, or the daily dose is reduced to 50 r or 75 r to each of the two fields, as soon as a pseudodiphtheritic membrane begins to appear. A thin membrane of this type usually disappears within a few days and the daily dose is then again increased to 100 r or 125 r to each field. One may often observe two such mild reactions —one about two weeks after the beginning of the treatment, the second toward the end of the series of exposures. A pseudodiphtheritic membrane did not appear at any time in 2 patients who have now been clinically free from cancer five and a half and nine and a half years respectively, since the last roentgen treatment.

Other technical factors have not been varied much during the course of the years, 200 kv., 8 to 25 ma., 1 to 2 mm. Cu filter, 50 cm. skin target distance, and 10 to 20 r/o per minute have been used routinely. We have gathered certain clinical impressions gained from repeated inspection of the irradiated tissues months and years after treatment. Thus, it seemed to us that for similar dosages and fields the reactions of the skin and mucous membranes were more severe if 0.5 mm. Cu was used as a filter than if 1 or 2 mm. Cu was used. These unfavorable tissue reactions are insignificant when compared to those due to the other factors discussed previously, i.e. large fields and intensive daily and total doses.

STATISTICS OF FAILURES

When the results in the 123 patients treated only by roentgen therapy are analyzed, one finds that 92 are dead, 8 are alive with disease and 23 are clinically free from cancer.

Patients Dead. Among the 92 patients classified as dead, there were 10 in whom there was no clinical evidence of cancer at the time of death; 4 of these patients had died of cardiovascular disease, one of tuberculosis of the lungs, one of pneumonia six years after treatment, one of acetanilid poisoning three years after treatment and 3 because of radionecrosis. Gross and microscopic examination at autopsy in these 3 radionecrotic cases did not disclose cancer, though positive biopsies had been previously obtained from the larynx. Death in these patients was probably due to an overdosage of roentgen radiation. In addition to these 10, there were 2 other patients in whom cancer could not be demonstrated locally at the time of death. One, however, had metastasis to the cervical lymph nodes and the other metastasis to the liver. From the standpoint of local control of the disease, it could be argued that these 12 cases should be added to those in which the treatment was successful!

Of the remaining 80 patients, cancer was known to persist in the larynx at the time of death in 47. Cancer may also have been

present in the other 33, but this was masked by marked edema and some necrosis. An autopsy was not performed on these cases. It therefore remains unknown whether this edema and necrosis were due to hidden unrecognized infiltration with cancer, or were the result of too large daily or total roentgen-ray dosages.

Untreated cancer of the larynx is usually a rapidly lethal disease and kills the patient within a year or two after initial symptoms become appreciable. The treatment in the 8 patients in Table II who are alive with clinical evidence of locally persisting cancer had been administered in 1937 and 1938. All patients treated longer than two years ago are either dead or well. Of the 92 patients classified as dead, the accurate date of death is known in 87. Of these 43 died within the first year after treatment, 32 within the second year, 3 within the third year, 3 within the fourth year and 6 within four to seven years. In other words, 89.6 per cent of the patients dying with persisting cancer of the larynx after failure of roentgen therapy died within three years of treatment.

Three Year Statistics. These findings suggest that three year statistics after roentgen therapy of cancer of the larynx are of greater importance than similar statistics in less rapidly lethal varieties of cancer. The status of patients treated more than three years ago and reviewed in July and August, 1940, as seen in Table IX is therefore significant.

TABLE IX

STATUS (AUGUST, 1940) OF 81 PATIENTS TREATED 1926–1936

Date Treated	Total	Dead	S.F.
1926–1931	14	14	
1932	9	6	3
1933	13	13	0
1934	13	10	3
1935	13	10	3
1936	19	16	3
Totals	81	69	12

From Table IX it will be noted that, except for the first period between 1926 and 1931, when chiefly advanced cases were treated and technique was poor, the most marked reduction in the number of clinical cures occurred in 1933. In view of the prognostic influence of the extent and location of laryngeal cancer, evaluation of the efficacy of any technique during any year must include considerations of these anatomic factors. For instance in 1933 there was an unusually high proportion of involvement of the arytenoids, 8 of the 13 failures showing this complication.

If the results of the years 1932 to 1935 are analyzed on anatomic basis, as seen in Table X, one obtains a fair picture of our effectiveness during that period. It is hoped that our more recent five year statistics will show an improvement. It will be recalled that the daily and total quantity of roentgen rays, the size of the fields and the reactions of the mucous membranes have been reduced in the past few years, while the period of treatment has been prolonged.

TABLE X

RESULTS OF ROENTGEN THERAPY IN PATIENTS TREATED, 1932–1935

	Total	Dead	A.D.	S.F.
Vocal cord	15	11		4
Band	4	3		1
Subglottic	1	1		
Epiglottis	14	11*		3
Aryepiglottic	1			1
Arytenoid	13	13		
Totals	48	39		9

* One patient died of pneumonia six years after treatment without evidence of cancer.

ANALYSIS OF FAILURES AFTER ROENTGEN THERAPY ALONE

A review of the 123 cases in which a single series of roentgen exposures was the only treatment discloses that the following local factors contributed to failure of the treatment: invasion of the arytenoid cartilage or base of the tongue; local extensiveness, especially if it required tracheotomy;

involvement of regional lymph nodes particularly in cord and arytenoid cases in contrast to band and epiglottic cases, several of which were cured in spite of regional metastases. Evaluation of the bearing which differentiation of the cancer may have on radiocurability cannot be deduced from the available material. Poor general health of the patient may interfere with the administration of proper roentgen-ray dosage. Technical causes of failure were underdosage (too small fields, too small dosage) or overdosage (too large fields, too intensive daily or total dosage) and dehydration. Of 48 patients treated by a single series of roentgen exposures, 9 are well over five years.

FAILURES AFTER COMBINED TREATMENT

The results of treatment in the 43 patients who received other therapy in addition to a single series of roentgen exposures were studied next. The cases were not analyzed in great detail as it was felt that the numbers in the various groups were too small to permit definite conclusions. They were arranged according to the type of treatment and the bearing this might have on survival. This analysis appears in Table XI.

TABLE XI

RESULTS IN 43 PATIENTS, ACCORDING TO TYPE OF TREATMENT

	Total	Dead	A.D.	S.F.
Roentgen therapy after previous irradiation with radium or roentgen rays	7	7		
Roentgen therapy after partial laryngectomy	12	4	4	4
Roentgen therapy after total laryngectomy	19	12	0	7
Pre-laryngectomy roentgen therapy	5	3	1	1

Repeated Irradiation. It is generally conceded that inadequate preceding local irradiation of cancer interferes with the success of subsequent radiotherapy in the same location. It is therefore not surprising that all of the 7 patients who had had previous irradiation died from cancer within a year after the more intensive treatment. These were second or third attempts to control the disease by irradiation and, in retrospect, seem futile except as a palliative measure.

Roentgen Irradiation after Partial Laryngectomy. The material at hand does not lend itself to proper evaluation of roentgen therapy after partial laryngectomy. In none of the 4 patients who appear clinically cured was there microscopic proof that cancer was present at the time when roentgen therapy was given. The remaining 8 cases are either dead or alive with disease.

The part of the thyroid cartilage remaining after partial laryngectomy has a poorer blood supply and lessened resistance to infection, to trauma and to irradiation. Exposure of this cartilage to intensive roentgen therapy may be followed by chondronecrosis, especially if an unhealed postoperative sinus leads to this cartilage and offers a ready pathway for the entrance of bacteria. Among the 12 who had partial laryngectomy and postoperative roentgen therapy, there were 3 cases of chondronecrosis.

Roentgen Irradiation after Total Laryngectomy. This treatment is indicated if there is microscopic evidence in the borders of the laryngectomy specimen that cancer has been cut across and left behind in the adjacent soft tissues. At times it controls the remaining disease, at others it fails to accomplish this aim. In 3 of the 6 patients in this group treated over six years ago who are now classed as "free from symptoms," microscopic evidence of cancer was present along the line of excision of the laryngectomy specimen, i.e., before the initiation of roentgen therapy. On the other hand, cancer persisted locally at the time of death, in spite of postoperative roentgen therapy in 8 of the 12 patients in this group who died. It has been emphasized previously that cures by roentgen therapy alone were accomplished only rarely in cancers invading the arytenoid cartilage; in these, surgical removal followed by roentgen therapy is at times indicated.

Recurrence in Tracheotomy Opening. Cancer was not controlled in a single instance in which the disease reappeared clinically in the wall of the tracheotomy opening. Roentgen therapy with 200 kv. in cancer of this group of cases has in our experience been of only temporary palliative value.

Pre-laryngectomy Roentgen Therapy. This form of treatment offers a clinical test of radiosensitivity permitting a change to surgery if the tumor proves radioresistant. The available information regarding the efficacy of this method is as yet very limited. In one patient who is now well three and a half years, laryngectomy was done as the disease persisted after roentgen therapy; in 2 of the 3 patients who died the roentgen-ray dose was too small to permit evaluation of the method.

ANALYSIS OF FAILURES AFTER "COMBINED TREATMENT"

Though definite conclusions cannot be drawn from this small series of 43 patients, it seems suggestive that (*a*) roentgen therapy failed in all patients in whom it represented a second or third attempt to control cancer by irradiation; (*b*) roentgen therapy after partial removal of laryngeal cartilage was accompanied by chondronecrosis in several instances; (*c*) 3 patients remain well six years or longer after the treatment whose (total) laryngectomy specimens showed that cancer had been left in the tissues adjacent to the line of excision.

SUMMARY

In summing up the findings of this study, it appears that our failures to control cancer of the larynx by roentgen therapy were due mainly to local peculiarities of the tumor and tumor bed or to faulty treatment. Reduction in the number of failures due to the first group of causes, we believe, must come through earlier diagnosis, permitting roentgen therapy before the cancer has become extensive. Improvement of failures due to the second category of factors will be obtained through avoidance of underdosage and overdosage.*

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* For discussion see page 32.

CANCER OF THE LARYNX

SURGICAL TREATMENT*

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SURGERY for cancer of the larynx should be limited to those cases that fulfill certain rigid requirements, namely:

1. The growth must be intrinsic.
2. It should be of a low grade malignancy.
3. There must be no adenopathy.

The Growth Must Be Intrinsic. Semon, years ago, pointed out that most laryngeal cancer starts as an intrinsic growth. Usually the primary site is the true vocal cord; frequently the anterior part of the cord. Originating on the vocal cord the lesion slowly infiltrates along the long axis of the cord and here it may remain localized for months or for years. The lymphatics in the anterior part of the larynx are few and scattered.

In the posterior part of the larynx the lymphatics are numerous, so the spread of cancer, when it reaches the posterior larynx, is rapid.

Unfortunately, many patients do not seek medical advice until the growth is far advanced. In the past ten years 214 patients with cancer of the larynx were seen at the Massachusetts Eye and Ear Infirmary. Of these only 72 were intrinsic.

When the disease has spread beyond the confines of the intrinsic larynx, surgery offers but little hope of cure.

Degree of Malignancy. Surgery of the larynx should be restricted to growths of adult cell characteristics. The greatest percentage of surgical cures are obtained when the growth is of Grade 1 or 2, according to Broders' classification. Imperatori agrees that the "histological grading should be of 1 or 2 of Broders' classification." However, Jackson and Jackson[1] state that "if the patient has a small cancer in the middle of the cord, and direct laryngoscopy shows that the lower edge of the growth grossly does not extend any further anteriorly than the anterior third, laryngofissure is the treatment of choice, no matter what may be the histologic estimate of the degree of malignancy." I do not agree with this statement.

When the growth is of a low grade it remains a surface lesion with but a minimum of infiltration into the stroma for a considerable period of time. The excision of a cancer of the vocal cord should be carried out through normal tissue at least 1 cm. from the margins of the lesion. The probability of being in normal tissue with a slow localized lesion is much better than when it is rapidly growing. When the lesion is of the anaplastic type, spread through the lymphatics is more extensive and rapid, and the probability of excising through normal tissue is greatly lessened.

Reviewing my cases of laryngofissure that developed recurrences, I find that of 6 cases, 5 were classified as Grade 2. There was only one recurrence of a Grade 1 carcinoma. From this experience I consider that the higher the grade, the greater is the surgical risk.

There Must Be No Adenopathy. Enlargement of the cervical glands contraindicates laryngeal surgery. The probability that metastasis has already taken place is so great that surgery of the larynx should not be the treatment of choice in the presence of enlarged cervical glands.

SURGERY OF THE LARYNX

The surgical procedures for cancer of the larynx may be roughly divided into two groups: first, that for small localized lesions, usually involving the anterior portion of

* Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

the larynx—the larynx splitting operation, or a modification of it; and second, that for extensive intrinsic lesions—total laryngectomy.

The choice of the operation suitable to use on a given case depends upon four factors:

1. The location of the growth.
2. The extent of the lesion.
3. The degree of malignancy.
4. The degree of cord fixation.

Laryngofissure. The indications for the surgical treatment of laryngeal cancer by laryngofissure vary but little with various operators. They all agree that the lesion should be small and limited to the anterior half of the larynx.

Beck and Guttman[2] state: "Laryngofissure is indicated when the neoplasm is small and restricted to one vocal cord. The disease must not have invaded either the anterior or the posterior boundary of the cord, and the cord must be mobile."

In the opinion of Imperatori[3] the operation is limited to "unilateral cordal growths located anterior to the vocal process and not much, if any, in the anterior commissure."

On the other hand, Jackson, Thomson, Tucker, and New state that the cause is amenable to laryngofissure even if the growth extends to the anterior commissure and involves the anterior portion of the other vocal cord—by using the Jackson anterior commissure technique.

Mobility of the Vocal Cord. Coutard[4] states "if the vocal cord is fixed, surgery and not radiation is indicated." Beck doubts that surgery should be used in the presence of cord fixation. Imperatori believes there should be little or no cord fixation. Immobilization of the vocal cord suggests the possibility of infiltration by the growth. In this case many speak of immobility of the cord as being due to chronic infection. This, I believe, is wishful thinking. To me immobility of the vocal cord in a lesion from which a biopsy has not been taken, in which there is no macroscopic evidence of infection, means infiltration by tumor.

In a follow-up of 39 laryngofissure operations, Thomson[5] finds that in 37 in which the cords were mobile, there were 31 five year cures, or 84 per cent. In 20 cases in which mobility was impaired, there were 15 cures, or 75 per cent. But when the cord was fixed in 9 cases, the cures numbered only 3, or 44 per cent.

Since Buck's first operation on the larynx for cancer in 1851, the larynx splitting procedure has become standardized for localized lesions of the anterior larynx. The operation of laryngofissure is performed under local anesthesia. A nasal feeding tube is passed preliminary to the operation, the thyroid cartilage and thyrohyoid membrane are split in the midline, the inner perichondrium of the involved side is dissected free from the inner surface of the thyroid cartilage throughout its whole extent, the tissue is removed with the diathermy cutting current, and the cut edges seared with the coagulating needle. A small tracheotomy tube is placed in the cricothyroid membrane as a safety valve and the larynx is closed in layers. The tracheotomy tube is removed in twenty-four to forty-eight hours. The nasal feeding tube is removed when the patient can swallow normally, usually in two to three days.

This operation has in its favor:

1. It is performed under local anesthesia.
2. Mortality is less than 1 per cent.
3. Hospitalization averages ten days to two weeks.
4. Unless the vocal process is removed, there is frequently a regeneration of the vocal cord.

When the case has been properly selected as suitable for laryngeal surgery by laryngofissure, three to five year cure can be obtained in approximately 75 to 82 per cent of cases, depending upon the operator.

Laryngectomy. In my opinion the operation of total laryngectomy should be advised if the lesion is

1. An intrinsic carcinoma of the larynx—of Grade 1 or 2, of sufficient extension to make its operative cure by laryngofissure questionable.

2. Involving the muscles or cartilage as evidenced by cord fixation or perforation of the cricothyroid membrane.

3. Subglottic carcinoma with cord fixation.

I would like to emphasize that the growth must be intrinsic. In 5 patients who developed recurrences after total laryngectomy, in all 5 I found involvement of the postcricoid region at the time of operation.

The tumor should be of Grade 1 or 2. In a review of the records of the Massachusetts Eye and Ear Infirmary since 1930, there is only one patient operated on in 1934, alive and well, who had a Grade 3 growth.

Coutard[4] states that in his experience "subglottic growths are radio resistant." When too extensive for cure by laryngofissure, total laryngectomy is indicated.

Surgery of the larynx for carcinoma should only be performed on patients who present a good or fair surgical risk. As with any major operation, arteriosclerosis, advanced diabetes, advanced pulmonary tuberculosis, complicate the recovery. Age is not a contraindication if the patient is a good physical risk. With our present methods of diabetic control, each patient must be evaluated individually before operation is advised. One of my eight year total laryngectomy patients was an advanced diabetic at the time of his operation. My last patient on whom I performed a total laryngectomy was seventy-four years of age and the recovery was the best of any in my series.

The mortality of total laryngectomy has been greatly reduced by the use of modern anesthetics. In my series of 35 cases there has been but one operative death.

When the patient has been properly selected, and by properly, let me repeat, an extensive intrinsic growth of adult cell type malignancy, without enlarged lymph nodes, the operative statistics will average three to five year cures in approximately 75 to 79 per cent of cases by total laryngectomy.

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DISCUSSION OF SYMPOSIUM ON THE DIAGNOSIS AND TREATMENT OF CARCINOMA OF THE LARYNX*

DR. GORDON E. RICHARDS, Toronto, Canada. The value of this symposium should be at once obvious to those who have listened to these very illuminating papers. Our own experience in the treatment of laryngeal cancer has been limited to 56 cases, of which only 21 have been truly intrinsic lesions of the larynx; however, we have this much to contribute: Up to 1933 our efforts were limited to radium, which included the fenestration operation and the interstitial application of radium within the larynx. There are no great technical difficulties in connection with either of these procedures but the results were disappointing.

From 1933 to 1938 our treatment of laryngeal cancer was almost entirely carried out by means of a radium bomb containing 4 grams of radium element and since 1938 by roentgen therapy at 400 kv.

Out of a large experience in the treatment of other types of oral cancer, we had reached the conclusion that in order to control squamous carcinoma, a dosage must be reached in excess of 5,000 r, delivered in the tumor. Personally, I believe that this can be delivered with greater ease and certainty and with fewer complications by means of roentgen rays at 400 kv. than at 200 kv. As Dr. Quick emphasized, infection is the greatest obstacle, whether it occurs prior to treatment or subsequently.

Dr. Lenz has pointed out that the greatest percentage of failures occurs when involvement is in the arytenoids, and this agrees with our

* Papers by Drs. Caulk, Quick, Lenz and Schall.

experience except that we would also include subglottic lesions. We have had at least 2 cases in which the arytenoids were involved, in which the disease disappeared or appeared to be under control.

I think everyone will admit that Dr. Schall's paper was a very fair presentation of the surgical side of the problem. The chief difficulty arises in limiting the surgeons to the precise definitions which he laid down as surgical possibilities. One must also draw attention to the fact that such a rigid restriction of the laryngofissure operation represents a very small percentage of the total cases of laryngeal cancer, and the radiologist is usually in the position of having to assume the responsibility for the rest. I am quoting from memory but I think a recent publication by Chevalier Jackson reports that in thirty-nine years he had 79 cases suitable for laryngofissure.

Our own experience is small, as I said. It indicates, however, that in those cases which would fall in the group which Dr. Schall would regard as suitable for laryngofissure, we have had 7 cases, of which 6 appear to be cured, which is a percentage of 85. Of those further advanced, we had 10 cases, of which 5 appear to be cured, or 50 per cent; and of the very far advanced, which would certainly not be surgical, 1 out of 4, or 25 per cent. This is an average of 60 per cent for the whole group.

I should like to record as my concluding contribution to these remarks the conviction that before proceeding to such a mutilating operation as a laryngectomy, a patient should be given the opportunity of a radiological cure. This would pick out all of the radiosensitive cases and, if the technique is carried out along the lines laid down by Dr. Quick, it will effectively control a very considerable number of patients who might otherwise be submitted to laryngectomy. The final proof in our own figures is that two of our best cases are doctors who quite definitely declined to have a laryngectomy.

Dr. Charles L. Martin, Dallas, Texas. First I want to congratulate both the officers of the Society and the essayists on the high quality of the papers presented in this symposium. The critical manner in which the material has been handled is especially commendable. My own experience, like that of Dr. Richards, is limited and I will only attempt to emphasize some of the points that have been mentioned.

The first slide shows a lateral view of the neck of an elderly patient treated successfully with roentgen rays seven months previously at the University of Michigan for a primary carcinoma of the arytenoids large enough to require a tracheotomy. Although he had been symptom free for six months we found it necessary to do a gastrostomy because of a complete occlusion of the esophagus produced by a postcricoid recurrence. At admission he had a pneumonitis of a patchy type which is frequently encountered in these cases. Although intensive therapy was given over the obstructed area this man died in about six weeks. Autopsy revealed a mass of necrotic tissue about the esophagus at the point of obstruction and little evidence of carcinoma. The bad prognosis of lesions of the arytenoids and postcricoid area has been generally recognized.

The danger of pulmonary infection in old men receiving heavy irradiation over the trachea has perhaps not been sufficiently emphasized. Roentgenograms of the chest should be made at intervals during treatment and the care of a good internist during the reaction stage is highly desirable. The next slide shows the patchy type of pneumonitis encountered in such a case. The temperature was not very high and the leukocyte count was only slightly elevated, but the patient did not recover. The third slide reveals a complication which is most unusual. A squamous cell carcinoma, Grade 3, on the vocal cord of a man thirty-two years old responded nicely to irradiation, but he soon developed a low grade temperature and a nonproductive cough and began to lose weight. When a thoracic surgeon opened the left thorax in an effort to drain a lesion which we had diagnosed as an abscess he discovered it to be a solitary metastasis so situated that it could not be resected.

No doubt most of the radiologists in the audience are principally interested in therapy technique. I visited Dr. Lenz some six years ago and observed his work. Soon afterwards I adopted a technique similar to his because it was much better suited to my clientele than the more involved method described by Dr. Quick. The factors used in 5 of my most successful cases are given in Table 1.

The disposition of cases which show a poor response to radiation therapy is a vexing prob-

TABLE I

CARCINOMA OF LARYNX

	Location	Pathology	Filter	Distance cm.	Dose	Area	Result
J.E.B.	Both cords	Epidermoid carcinoma No Grade	0.5 mm. Cu+1 mm. Al	50	300 r×20	10 cm.	Well 6 yr.
F.P.	One ventricle	Epidermoid carcinoma Grade 2	2.0 mm. Cu+1 mm. Al	50	300 r×20	7 cm.	Well 5 yr.
G.W.B.	One cord and arytenoid	Epidermoid carcinoma Grade 3	2.0 mm. Cu+1 mm. Al	50	300 r×20	7 cm.	Well 5 yr.
W.S.W.	One cord	Epidermoid carcinoma Grade 2	2.0 mm. Cu+1 mm. Al	50	300 r×20	7 cm.	Well 5 yr.
P.S.	Both cords	Epidermoid carcinoma No grade	Thoraeus	50	300 r×20	7 cm.	Well 4 yr.

lem and I hope that Dr. Schall will tell us whether or not they may be safely operated upon. If so, it seems to me that we may now feel free to advise all patients with intrinsic carcinoma not involving the arytenoids and not subglottic in origin to try a properly planned series of roentgen treatments before considering surgery.

Dr. H. W. Jacox, Pittsburgh, Pa. I should like to ask Dr. Lenz how he explains the fact that from 1926 to 1931 there were 15 patients referred to his department whereas in other years, from 1932 on, he had that many or more per year. I think that is an important point and gives us some indication of what the laryngologists are doing and what they think about it.

Dr. John T. Murphy, Toledo, Ohio. I am glad that Dr. Martin emphasized some of the complications because I don't think that there was quite enough said about complications in the papers. If you are going to use the technique that Dr. Quick and Dr. Lenz advocate, you must be prepared for complications that come on during and after you finish your treatment. I have had 2 cases which developed acute infections of the upper respiratory tract, one during the latter portion of the treatment, and one six months afterwards, causing the entire irradiated field to slough out. This has happened to other people than myself so if you are going to use this heavy treatment you must be prepared to face what may happen later on.

Dr. William Harris, New York. The question of surgery following roentgen therapy of cancer of the larynx has been brought up in the discussion. There is no doubt that there was considerable danger in doing extirpative surgery following roentgen therapy such as was done in 1931 and for several years thereafter. This was due to the fact that large portals of entry were used and, in many cases, the treatment was given too quickly and there was considerable compromise of the blood supply in the tissues surrounding the larynx. In recent years, smaller portals of entry have been used, thus diminishing the damage to the nutrition of the tissues around the larynx. This has made it possible to do extirpative surgery successfully. In the past three years, total laryngectomies were done in 5 cases following unsuccessful roentgen therapy for cancer of the larynx. There was good healing and there were no unusual complications.

The points brought out by Dr. Quick and Dr. Lenz should always be kept in mind, that is that meticulous care should be exercised in

the treatment of cancer of the larynx, such as the choice of fields, filter and variations in treatment to suit the individual needs of the patient.

We have stopped using the anterior fields except in anterior commissure disease, because we feel that if surgery is to be done later, the incision is very apt to pass through this point and might interfere with healing.

Dr. Quick (closing). There are just two or three points I would like to emphasize in closing. First is the very limited anatomic zone we must keep in mind in a discussion of this sort, namely, the laryngeal box or the intrinsic larynx proper. When we get outside that zone, we are strictly away from the type of growth suitable at all for surgical procedure. Apart from that, the epiglottis, for instance, is a problem by itself. It is not strictly a laryngeal problem. The pyriform sinus and the region at the base of the tongue are hypopharyngeal problems and not strictly laryngeal. The postcricoid area belongs to the esophagus rather than the larynx.

I should like to emphasize again the attention to the detail of technique. I probably overdid it in some respects in my paper and I don't mean that the various details that I laid down there are going to apply indefinitely. They will be changed but at the present time, from our experience, we believe they offer the best chance.

Now, I think it is paying attention to these technical details right straight along that permits of satisfactory results. It is slow, it is tedious, it is time-consuming—it is all of that—but, if we are going to cure the largest percentage of these cases, then we must pay attention to those things.

For instance, I thoroughly agree with Dr. Martin that the factors which he gave us will cure a very substantial number of these patients. I do seriously think, however, that if we are going to cure the maximum number by irradiation, probably a higher dosage and more strenuous factors are necessary and I do think with the lower roentgens per minute, we will cure more patients, and have more people with more nearly normal tissues as final results. I refer to dryness of the membrane, which is distressing to these patients afterwards, and the minimum damage we do to the normal structure is ultimately important.

I had one patient treated by smaller factors than I have suggested today, who, whenever he gets a very severe head cold, is likely to develop a laryngitis, and there is a period of a fortnight when he has to be watched carefully. He almost got in trouble one time through secondary necrosis in the membranes. Now, that would not occur, or has not at least, with the more tedious factors that I suggested, and I think by paying attention to the daily care of these patients while they are under treatment by irradiation, keeping them sprayed with oil sprays and seeing that their nasal breathing apparatus is clear and wide open, so that they sleep breathing through the nose and not through the mouth, and drying the membrane unnecessarily from day to day—I think that, plus knowing where to stop with the top dose, takes care of many of the complications such as Dr. Murphy mentioned.

I see no reason for having gross necrosis of the mucosa from irradiation if we know where to stop, if we take good care of the patients along the way.

Finally, I should like to emphasize what I regard as a most important factor. Infection and not histology is the controlling influence in the response of these patients to irradiation.

Dr. Lenz (closing). As Dr. Jacox surmised the small number of cases recorded between 1926 and 1931 was due to lack of confidence of local laryngologists in the efficacy of roentgen therapy in cancer of the larynx. The number of referred patients increased greatly after we had proved the value of this treatment to the satisfaction of the late Dr. Coakley, of the present laryngological staff of the Presbyterian Hospital and of some of the men at the Manhattan Eye, Ear and Throat Hospital.

Dr. Quick who has limited his discussion to what is "usually referred to as intrinsic larynx" believes that "anatomically and as a clinical cancer problem, the epiglottis stands as a separate entity." While there are unquestionably differences in the behavior of these two groups of cancer, I cannot agree with this view. I prefer to interpret the anatomic extent of the organ somewhat more broadly than suggested by Dr. Quick and wish to include cancer of the epiglottis in a discussion of cancer of the larynx. In the printed paper, however, I will exclude the cases of cancer of the pyriform sinus which should not have been included in this report.

Radionecrosis of the laryngeal cartilages referred to by Dr. Murphy was frequent in our

early experience, but fortunately has occurred rarely in recent years. Though most of these cases were complicated by the presence of cancer, this was absent at autopsy in at least three instances. These cartilages had been exposed to too great daily or total quantities of roentgen rays and necrosed when bacteria entered these poorly vascularized structures.

In the early days of roentgen therapy of cancer of the larynx aspiration bronchopneumonia was not uncommon especially in debilitated patients. They had usually become dehydrated as they stopped drinking because of the dysphagia which followed the irradiation reaction in the lower pharynx. More recently this pulmonary complication has occurred rarely. The reactions produced in the pharynx are milder, patients are hospitalized during and immediately after the height of the reaction in the mucous membranes, and dehydration is combatted by gavage, procto-, hypodermo-, and veno-clysis.

Hematogenous metastases to the lungs are rare in cancer of the larynx as emphasized by Dr. C. L. Martin. This did occur in at least one case in our series. Repair of laryngectomy wounds has not been bad where the operation followed a complete course of roentgen therapy. The intensity of the treatment and the size of the field have influenced the healing, as brought out by Dr. Harris. In a case of cancer of the larynx in which roentgen therapy had failed, Dr. Imperatori did a successful laryngectomy. There was only a short delay in healing of the wound, which has now remained healed three and a half years.

Finally, I would like to urge Dr. Schall to consider laryngectomy followed by roentgen therapy in differentiated intrinsic cancers of the larynx which have invaded the arytenoid cartilages or extended beyond these. These are cases which ordinarily do not do well with either surgery or irradiation alone. Three such cases in our series treated in this manner have now been free from clinical evidences of cancer for years.

PRESIDENT SOSMAN. Before I call on Dr. Schall for the closing discussion, I should like to emphasize the thing he was apparently surprised to find out, and that is that we did not invite a surgeon here to make a Roman holiday out of his display of poor results or his attitude, but we invited him here to cooperate with us, and I think that is the proper attitude of all radiotherapists, perfect cooperation and coordination of efforts. Dr. Schall, will you close?

DR. SCHALL (closing). Thank you, Dr. Sosman. You took the wind out of my sails because you said what I was going to say. I laid down what I think are rather rigid rules for surgery, and I had hoped to make a plea for close cooperation between the surgeon and the radiologist because I know that by just such close cooperation can we really do the best for our patients.

In answer to some of the questions, I have had no experience with surgery after irradiation has failed.

In reference to cricoid carcinomas, they have been almost 100 per cent failures. I have one patient with an adenocarcinoma alive and apparently well three years after irradiation by Dr. Hampton.

I thank you for your kind reception of my contribution. I believe, let me repeat, close cooperation is a necessity, and only by close cooperation between the surgeon and the radiologist can we obtain for our patients the best results.

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DIFFICULTIES IN DIFFERENTIATING MIDBRAIN LESIONS FROM CEREBELLAR LESIONS*

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THERE seems to be a general tendency, when selecting material for medical publications, to choose cases which are either extremely rare and therefore interesting, or cases in which the author has been fairly successful with his methods of diagnosis or treatment. Such a tendency is particularly noticeable in the literature that deals with the diagnosis and localization of brain tumors. Very little emphasis has been placed upon the difficulties of clinical and roentgenological cerebral localization, even with the best of techniques, hence leaving the erroneous conception that such difficulties in the hands of well trained individuals are minimal. Certainly to anyone dealing with a large group of tumors, difficulties in diagnosis arise often enough to remind us of the inadequacy of our methods.

In the radiological literature one is amazed by the high degree of accuracy with which brain tumors are localized by roentgenological methods alone. When ventriculography and encephalography are used in addition to the usual films of the skull, brain tumors have been localized in as high as 95 per cent of the cases. In our clinic we appear unable to achieve such accuracy in brain tumor localization, especially if one considers consecutive unselected cases. It would appear that much benefit could be derived from a report of some of the pitfalls and difficulties in brain tumor localization even with complete roentgenological studies. For this reason we wish to report a small group of somewhat similar cases in which all methods of clinical investigation and careful roentgenological study have failed to accurately localize the lesion.

Although difficulties may be encountered in the localization of tumors in almost any part of the brain, the present discussion will be limited to those lesions involving the aqueduct of Sylvius and the cerebellum. These cases have been grouped together because of the great similarity in the picture presented by ventriculography. It is obvious that some of these lesions can be localized roentgenologically with a high degree of certainty. In others, the history and neurological findings alone, or in conjunction with the ventriculograms, are characteristic enough to warrant an accurate specific diagnosis of midbrain or cerebellar lesion. However, there still remains a significant number of cases which, in spite of all the methods usually employed, are difficult to localize definitely.

In the cases to be presented the roentgenograms show the following findings. The lateral ventricles are dilated, the degree of dilatation depending upon the extent and duration of the obstruction. There is no displacement of the lateral or third ventricles. The third ventricle and the anterior portion of the aqueduct are usually dilated although in a few instances, possibly because of inadequate filling with gas, this enlargement does not reveal itself in the ventriculograms. The fourth ventricle is also not filled. An obstruction located at any point posterior to the third ventricle is compatible with these findings. We have been unsuccessful in attempting a more accurate localization.

The question might be raised as to the practical importance of localizing a lesion in the midbrain as contrasted to one in the posterior fossa. Such a query can be answered by considering the indications for and routes of surgical approach. If the process is an invasive growth surrounding or invading the aqueduct, direct surgery to

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the local lesion is not attempted. However, it is often possible to prolong the patient's life and comfort by making an opening in the floor of the third ventricle and allowing the ventricular fluid to escape into the basilar cisterns, thus relieving the intraventricular tension. On the other hand, tumors of the pineal gland or cerebellum may be directly attacked by means of surgery. These tumors, however, may produce clinical and roentgenological pictures very similar to midbrain lesions, yet the surgical approach is entirely different. Surgery in either of these two areas is precarious and consequently, unless the surgeon is fairly certain of the exact localization, surgical intervention becomes too hazardous. Likewise a tumor occupying the superior anterior part of the cerebellum is attacked surgically in a different manner from a tumor located near the cisterna magna. Consequently it is readily apparent that, for purposes of therapy, it is extremely important to be able to accurately localize these various lesions.

We do not wish to infer that most lesions obstructing the aqueduct or fourth ventricle are difficult to identify. There are three types of ventriculographic findings that readily allow an accurate localization.

1. Cerebellar tumors situated inferiorly may produce a dilatation of the aqueduct as well as the anterior part of the fourth ventricle, and in many cases these areas are filled with gas. The caudal portion of the fourth ventricle is usually not filled, however, because of compression or invasion by the tumor. In the posteroanterior view the aqueduct and upper part of the fourth ventricle may be displaced away from the midline permitting further localization to the right or left cerebellar hemisphere. An example of such a case is as follows:

Case 1. K. M. was a white female, aged twenty-seven, whose illness began about one year prior to admission at which time she first noticed periodic headache. These continued to increase in severity so that in about four months they had become almost constant and were localized to the frontal and occipital regions. In the following month she first noticed the beginning of a definite diplopia and some blurring of vision which continued to increase until the time of her admission. There was an occasional attack of nausea and vomiting with the increasing severity of her headache. At the time of ad-

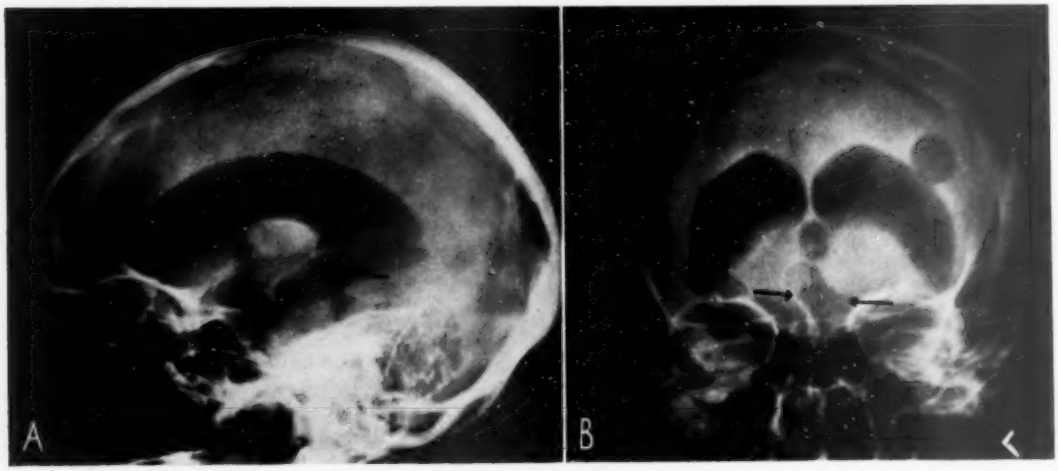

Fig. 1. Case 1. The lateral and third ventricles are dilated but not displaced. The aqueduct is clearly shown in the lateral view (arrow), *A*, and is dilated. The fourth ventricle is also visible in both the lateral and posteroanterior views and appears to be obstructed inferiorly. In the posteroanterior view, *B*, the fourth ventricle is also seen to be in the midline (arrows). A simple cyst was found protruding into the cisterna magna from the left side. In this type of ventriculogram it is not difficult to accurately localize the obstruction to the inferior part of the fourth ventricle.

mission the examination revealed a well developed female who was apparently suffering from a very severe headache, diplopia and visual impairment. Ophthalmoscopic examination showed a bilateral papilledema of about six diopters. There was also a paresis of the right sixth cranial nerve. The knee jerks were slightly more active on the left, although all the deep reflexes were hyperactive. The rest of the neurological examination was negative. It was concluded that the patient was suffering from a cerebral neoplasm, but the exact location was uncertain because of the paucity of clinical findings.

Ventriculography (Fig. 1). There is a symmetrical dilatation of the lateral and third ventricles as well as of the aqueduct. The fourth ventricle is well filled and is evidently obstructed in its most caudal portion and not displaced to either side. A cerebellar exploration was done and revealed a simple cyst in the left cerebellar hemisphere bulging into the cisterna magna. No tumor tissue was found associated with the cyst. The cyst was evacuated and its membrane was removed. Recovery was uneventful and the patient has been free of symptoms for three years.

Comment. Although a clinical localization was not possible in this case the ventriculographic findings were definitely those of a cerebellar lesion. Furthermore because the entire aqueduct and most of the fourth ventricle were so clearly demonstrated the pathology would have to be in the region of the cisterna magna. Also the lesion would probably be small and amenable to surgery.

2. A slow growing tumor within the fourth ventricle sometimes results in a gradual expansion of the ventricle. In these cases the injected gas may surround part of the tumor, often making it possible to actually see the mass itself within the cavity of the dilated ventricle. We have observed one case in which such roentgen changes were present but have not had the opportunity to verify these findings. The patient is still alive and consequently we cannot present it as a verified case.

3. Tumors of the pineal obstruct the aqueduct and often show a mass protruding into the third ventricle, obliterating the suprapineal recess. Since the clinical and roentgenological picture is usually quite clear an example will not be cited.

However, there still remains a small group of cases with involvement of these same regions in which one is unable to determine the accurate position of the lesion even by means of ventriculography. As stated previously, in such cases the ventriculograms show a symmetrical dilatation of the lateral and third ventricles without any displacement. A short segment of the anterior part of the aqueduct may be dilated and filled with gas. Often there is no gas whatever either within the aqueduct or the fourth ventricle. For a number of years it had been our custom to report these cases as posterior fossa obstructions, having vaguely in mind a cerebellar tumor which could be reached by a suboccipital approach. Frequent negative cerebellar explorations and postmortem examinations have convinced us that such roentgen pictures can be produced by variously placed lesions of the midbrain and cerebellum and that we are at present in no position to state the exact location. We can only conclude that the obstruction is posterior to the anterior part of the aqueduct, realizing that the actual pathology in any such case may be in or around the aqueduct, in the cerebellum or fourth ventricle, or around the foramina of Luschka and Magendie.

We have selected for report a group of illustrative cases and believe a brief review of their histories, clinical findings and roentgen investigations will illustrate the difficulties that one encounters in localizing tumors in this region. All the cases have been verified either by operation or autopsy. The mortality in these cases may seem high, but this is accounted for by the facts that only proved cases are included and that the region being considered is not readily amenable to treatment.

The first 3 cases are of cerebellar neoplasms. Usually one encounters little difficulty in identifying a cerebellar tumor following contrast studies. Occasionally however, for some unexplained reason, the

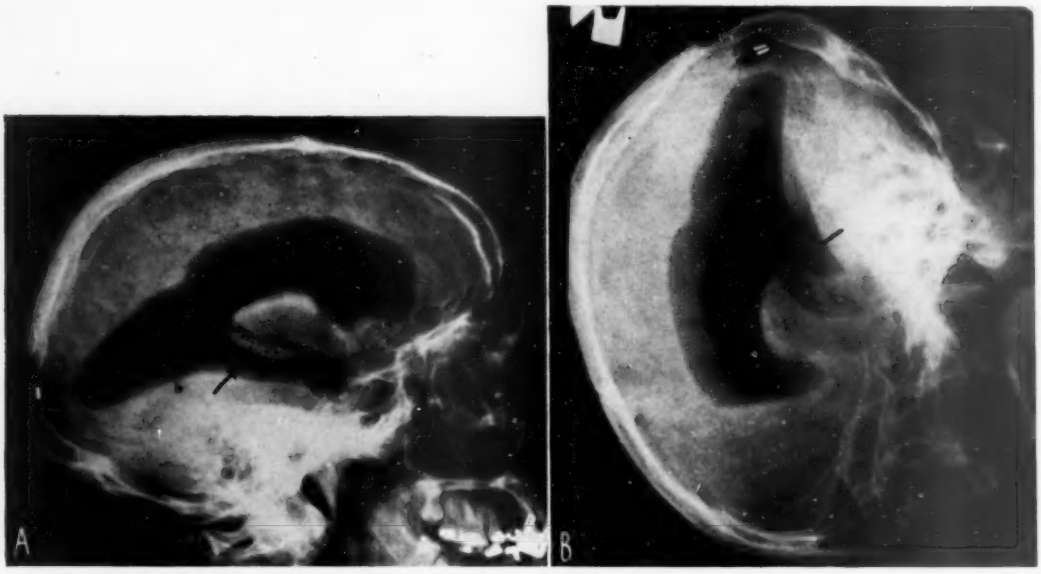

Fig. 2. Case II. There is a symmetrical dilatation of the lateral and third ventricles without displacement. The dotted line represents the posterior and inferior margins of the third ventricle and the demonstrable portion of the aqueduct (arrow). In the lateral view with the forehead down, *B*, it is obvious that the ventricles have been almost completely drained of fluid and consequently the lack of air filling of the aqueduct cannot be attributed to an inadequate quantity of air injected. Postmortem examination of the brain showed a medulloblastoma which filled the fourth ventricle but the aqueduct was dilated and not involved by tumor even though it did not fill with air.

dilated aqueduct fails to fill completely with air. One then obtains the erroneous opinion that the obstructing lesion is anterior to the cerebellum, probably in the region of the midbrain.

Case II. G. H., aged eighteen, six months prior to admission to the hospital began to have vague gastrointestinal symptoms manifested primarily in mild nausea, occasional vomiting, and a gradual loss of weight in spite of a good appetite. These symptoms continued for a period of three months. He then developed severe headache, primarily in the occipital region. Associated with the cephalalgia the patient also noticed a diminution of visual acuity and occasional attacks of dizziness and unsteadiness in gait. He began to deviate toward the right while walking, and his gait became very ataxic. A few days prior to admission, his vertigo became accentuated and he developed a definite diplopia.

Neurologic examination revealed a bilateral choked disc of two diopters. His visual fields were bilaterally constricted. There was a paresis of both sixth cranial nerves, a paresis of the lower right seventh cranial nerve, and a deviation of the uvula toward the right on phonation. A nystagmus was present. The right knee jerk was slightly increased over the left. There was a definite staggering gait with a positive Romberg sign. The laboratory examinations were all negative. The clinical impresssion was that the patient was suffering from a left cerebellar tumor. He was transferred to the neurosurgical service where a ventriculogram was made.

Ventriculography (Fig. 2). There is a symmetrical dilatation without displacement of the lateral and third ventricles. Only the upper portion of the aqueduct is filled with air. It was concluded from these ventriculograms that the lesion was either in the midbrain or in the cerebellum. Because the clinical findings were quite definitely in favor of a cerebellar lesion, a suboccipital craniotomy was performed and a tumor was exposed within the cerebellum. No attempt was made to remove the tumor. The patient died thirty-six hours later. The autopsy revealed a medulloblastoma filling the fourth ventricle and invading much of the floor of the rhomboid fossa as well as the adjacent medullary tissues. The aqueduct was not involved with tumor and was dilated.

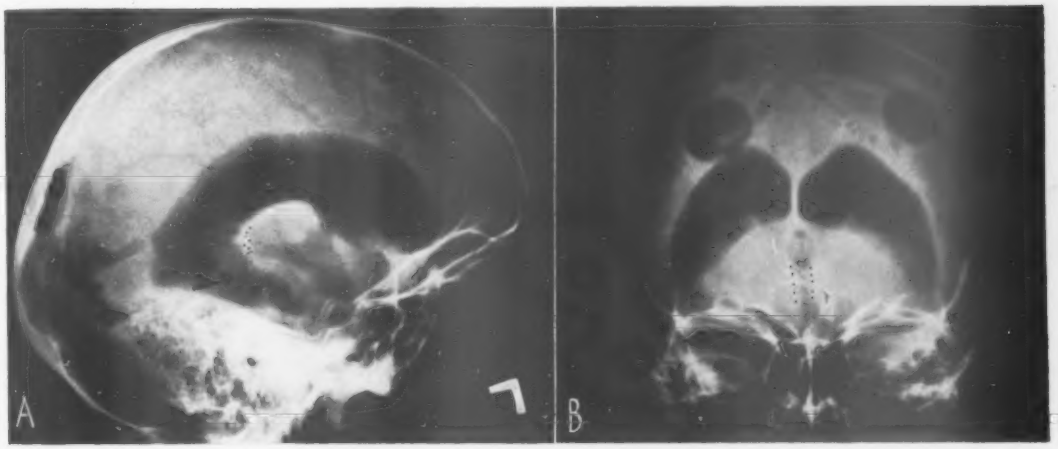

Fig. 3. Case III. Only the upper half of the aqueduct is filled. Its outline is indicated by dots in both *A* and *B*. A midbrain lesion might be expected. However, at operation a very small tumor of the right cerebellar tonsil was found and proved to be an angioblastoma. The tumor was so small it could not be expected to occlude the aqueduct at the level shown on the roentgenograms. One must conclude that the aqueduct was patent but did not fill completely with the gas.

Case III. E. D., white female, aged thirty-eight. Sixteen years prior to her entrance to the hospital she first began to complain of occasional dizzy spells which would persist for a very short time. About ten years later she began to have occasional occipital headaches which were at first periodic but became more and more constant. During this same period she also developed some diminution in her vision. About eight months prior to admission she first complained of diplopia, blurring of vision and some nausea. These symptoms continued more or less constantly. Associated with the nausea there was an occasional attack of vomiting which, according to the family, had been projectile in character. About one month before admission the patient's symptoms all became more intense. Her headache became very severe with the pain radiating from the occipital region into the neck and associated with dizziness and diplopia. She began to have severe attacks of vomiting which were projectile in type. She also had fleeting attacks of confusion, which lasted just a few minutes.

On admission examination showed bilateral choked discs of about four diopters with numerous retinal hemorrhages and some exudate. There was a left abducens paralysis. She had a marked diplopia, the left lid slit was wider than the right, and the vision was almost entirely destroyed. The deep reflexes in the upper limbs were normal. The deep reflexes in the lower extremities were hyperactive with the right knee jerk being slightly more active than the left. The Babinski sign was negative bilaterally. Coordination was good. It was suspected that the patient had a brain tumor, but the location of this tumor could not be determined.

Ventriculography (Fig. 3). The lateral and third ventricles are dilated. The upper part of the aqueduct is also dilated and appeared to be obstructed as it entered the fourth ventricle. These pictures were consistent with a lesion in the upper part of the fourth ventricle or the lower portion of the midbrain. Hence an inoperable tumor of the midbrain could not be excluded roentgenologically.

A cerebellar exploration was done with the hope that an accessible tumor would be encountered. Fortunately a tumor was found in the right tonsil, but this could not be predicted with certainty from the ventriculograms. It was well encapsulated and was rather small. The tumor was removed and proved to be an angioblastoma.

Case IV. J. H., white male, aged seventeen. Six weeks prior to admission the patient developed an acute infectious process, which was diagnosed as influenza. During this illness his temperature was moderately elevated and he suffered from diarrhea and vomiting which persisted for about three to four days. This condition gradually subsided. Three weeks later he developed a mild frontal headache and some nausea and vomiting. He was admitted to a

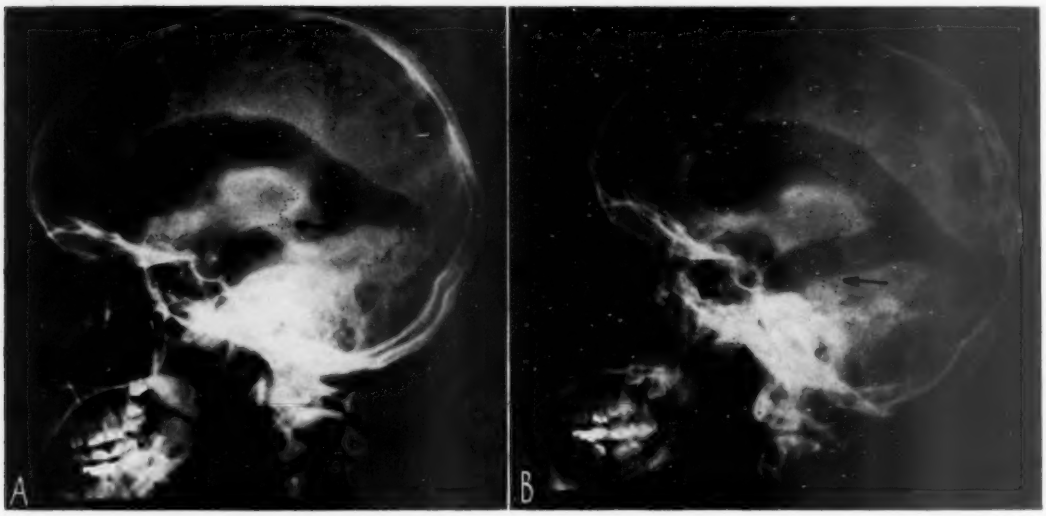

FIG. 4. Case IV. In this case our usual number of lateral views were made. *A* is part af the right lateral stereoscopic view and only a small part of the aqueduct is visible (arrow). In *B*, which is part of the left lateral stereoscopic view, the entire aqueduct was visible and was considerably dilated and displaced forward slightly (arrow). The lateral view made with the forehead down did not show any filling of the aqueduct. This illustrates clearly that the aqueduct at times may not fill with gas even though it is open and even dilated. Postmortem studies showed a large ependymoma involving the cerebellar hemispheres and also filling the fourth ventricle. The entire aqueduct was dilated.

local hospital where numerous spinal taps were performed. At this time he had no elevation of temperature but the headaches and vertigo persisted. The spinal taps showed only an increased spinal fluid pressure.

Examination on admission revealed a thin, dehydrated, lethargic, white male. His pupils were dilated. The left pupil was sluggish but reacted to light. The right was fixed and did not react. The disc margins were blurred, the right showing a three diopter elevation. The rest of the cranial nerves were negative. His deep reflexes in the upper limbs were more active on the right than on the left. The left knee jerk was slightly more active than the right. Coordination was fair in spite of his severe dizziness although he did have some difficulty with the finer movements of his hands. The clinical diagnosis on admission was encephalitis or a brain tumor. After several days of observation it was the opinion of the neurologic staff that the patient probably had a posterior fossa neoplasm. A spinal tap was performed and showed the pressure to be 40 mm. of mercury with four cells in the spinal fluid. He was therefore transferred to the neurosurgical service for air studies.

Ventriculography (Fig. 4). There is a symmetrical dilatation of the lateral and third ventricles and of the entire aqueduct. The aqueduct is also displaced slightly forward. These findings indicated an obstruction in the body of the fourth ventricle or below. A midbrain obstruction could be excluded. All of the lateral views, except one, however, showed only the proximal portion of the aqueduct to be filled with air. Had not this single film been obtained with the entire aqueduct filled, the same difficulty in localization would have been encountered as illustrated in the previous cases. This case illustrates clearly that the aqueduct may not fill even though there is no closure or obstruction. However, even though a midbrain lesion can be excluded, it is still not possible to decide whether the tumor is in the superior or inferior part of the cerebellum.

A cerebellar exploration was performed. The fourth ventricle and aqueduct could not be exposed because of the marked increased pressure and herniation which occurred. No tumor was found. The patient died twenty-four hours after the operation.

Autopsy Findings. A large tumor mass was situated in the superior surface of the cerebellum involving the right side more than the left. The aqueduct was markedly dilated in its first 1.5 cm. but then became compressed by the tumor, as it entered the fourth ventricle. The tu-

mor was soft, somewhat invasive and on section proved to be an ependymoma.

Comment. Cases II and III resemble midbrain obstructions roentgenologically when actually small cerebellar tumors were present in the region of the cisterna magna. Case IV illustrates that the aqueduct may or may not be filled with gas even though it is not obstructed. It also illustrates the difficulty of determining whether the tumor is located in the superior or inferior part of the cerebellum.

In the next 3 cases, the ventriculographic pictures resembled in many details those just described. Here, however, the lesions were all in the region of the aqueduct. A comparison of these two groups of cases readily illustrates the difficulty in differentiating by air studies lesions of the cerebellum from those within the midbrain.

Case V. C. L., a white male, aged thirty-two, became ill about fifteen months prior to his admission to the hospital at which time he fell out of a truck and was rendered unconscious. There was no headache associated with this experience. Following this incident he began to develop progressive visual impairment. Five changes of glasses did not offer him any relief or correction for his vision. He began to feel weak and was finally forced to discontinue his farm work. He began to manifest a progressive impairment of memory and difficulty in concentrating. Coincidental with these symptoms he also developed frequent attacks of unconsciousness which would occur daily and last from five to ten minutes. These attacks were preceded by a dizziness, weakness and generalized tremor of all muscles but no actual convulsive movements. During the past few months he developed a sexual impotence, a mild headache, especially over the frontal region, and a definite personality disorder. He became drowsy and sluggish, slept much more than usual, and showed definite signs of irritability and temper tantrums. There was a definite inclination to impulsive acts; at one time he pushed his fist through the windshield of a car without the slightest provocation.

On admission his blood pressure was 120/80. There was a bilateral choked disc of about one diopter, with a beginning secondary optic atrophy. He had a rapid nystagmus of low amplitude on the lateral gaze to either side. There was a questionable hyperesthesia over the right side of the face. The deep reflexes were increased on the left; abdominals were equal and active. There was a definite adiadokokinesis of the left arm and some edema of the eyelids. No definite diagnosis as to the location of the lesion could be made. It was believed that the patient had a brain tumor and that ventriculography was indicated.

Ventriculography (Fig. 5). The lateral and third ventricles are markedly dilated. The suprapineal recess is unusually large as is also the posterior inferior part of the third ventricle. The upper 1 or 2 mm. of the aqueduct is visible. There was no air in the rest of the aqueduct or fourth ventricle. From these films it seemed impossible to definitely conclude as to whether the obstruction was in the aqueduct or lower down in the cerebellum.

In view of the absence of a good cerebellar localization clinically, a right frontal craniotomy was done and the floor of the third ventricle perforated to permit cerebral spinal fluid to escape into the subarachnoid space and thus relieve the hydrocephalus. The patient made an excellent convalescence, his papilledema decreased, he became negative neurologically, and he was discharged from the hospital about one month after his first admission. It was believed at this time that the internal hydrocephalus was due either to some sort of infection such as arachnoiditis at the outlet of the fourth ventricle or the presence of a tumor along the aqueduct. The patient was again seen six weeks later in the outpatient clinic. He reported that his eyesight had improved and that he felt stronger and his appetite was better. Examination revealed a bilateral choked disc with a secondary optic atrophy. His pupils were small and reacted only slightly to light. There was constant movement of the eyes but no true nystagmus. The patient had some weakness on protrusion of the tongue. The reflexes were generally suppressed. Abdominal reflexes were normal. Sensation was grossly normal and there was a bilateral intention tremor. He continued unchanged for about two months at which time he suddenly became very much confused and incoherent. A few days later he became agitated, pounded on the walls of his room and tore up his bed clothes. He had recurrent generalized convulsions with loss of consciousness. He was brought to the hospital in an

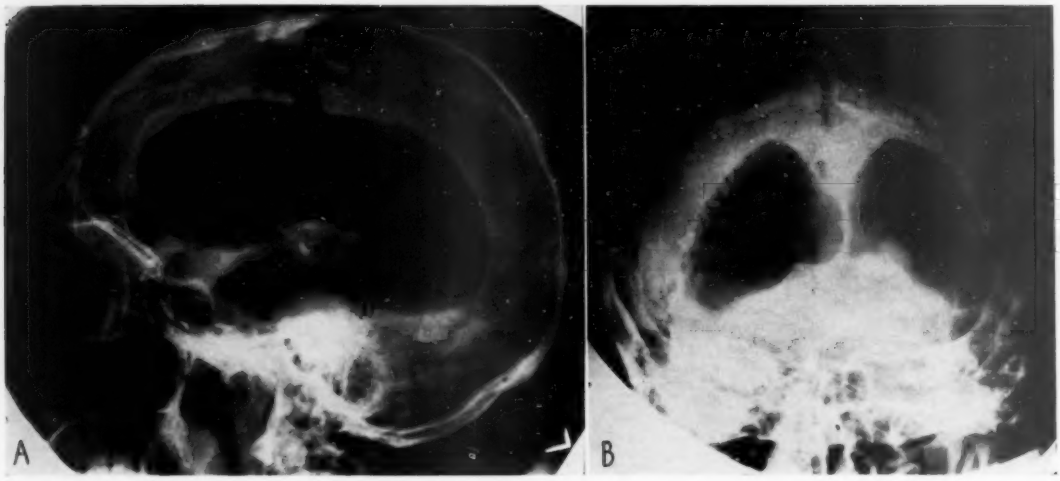

Fig. 5. Case v. The lateral and third ventricles are markedly dilated. In *A*, the suprapineal recess is extremely large (*1*). Only the upper end of the aqueduct contains gas (*2*). It is not possible to decide whether this represents a cerebellar tumor or a midbrain obstruction. The study was repeated about two months later and showed the same findings. At necropsy an obstruction 3 mm. in length was discovered in the midportion of the aqueduct. It was caused by an inflammatory gliosis.

uncooperative, confused state. Examination revealed a definite secondary optic atrophy with only a very small elevation of the discs. The rest of the cranial nerves, his reflexes and sensation were all within normal limits. In walking the patient tended to tip backward and was unable to perform the Romberg test. Findings now suggested a midline cerebellar lesion and a cerebellar exploration was performed. No tumor was found. Two days after this procedure the patient became stuporous and died.

Autopsy. Section through the mesencephalon revealed an area of constriction in the midportion of the aqueduct measuring about 3 mm. Microscopic study through this region showed an almost complete occlusion of the aqueduct by glial tissue. This was a strictly localized gliosis. Numerous round cells surrounded some of the adjacent vessels, suggesting an inflammatory reaction. It appeared, therefore, that the occlusion of the aqueduct was the result of an old inflammatory reaction with a secondary gliosis. Had we been able to state from the ventriculograms that the obstruction was definitely around the aqueduct the cerebellar exploration could have been avoided.

Case vi. H. O., a white male, aged thirty, whose illness dated back about three years, at which time he started to show signs of incoordination. This manifested itself primarily in clumsiness in performing rather delicate tasks with which he formerly had had no trouble. For about six months prior to admission the patient had shown definite signs of an unsteady gait. At about this same time, he had his first convulsive seizure which was of a generalized type and was associated with headaches which persisted for days afterwards. These seizures gradually increased in frequency so that for a short period prior to his admission he had as many as six a day. They would occur at any time during the day or night and would last from a few minutes to almost an hour. There also occurred a definite personality disturbance. Since the onset of his acute illness, the patient had become dull, morose, forgetful, and careless in his behavior and in his speech. He occasionally manifested definite signs of confusion and disorientation. On admission he appeared well developed and well nourished. He seemed somewhat clouded mentally and had a little trouble with coordination during the examination. There was a vertical nystagmus on looking upward, and at times on looking laterally in either direction. There was questionable left lower facial paresis but otherwise the cranial nerves were negative. There were no choked discs. The deep reflexes were suppressed and appeared to be less active on the right side than on the left. Babinski sign was positive on the right and negative on the left. The rest of the neurologic examination was conventional. The history and the neurological examination did not allow for any definite localiz-

ing diagnosis, although it was suspected that the patient probably had a supratentorial tumor. It was believed that air studies were indicated to further localize the lesion.

Encephalography and Ventriculography (Fig. 6). Encephalography was done first and this showed only a small amount of air in greatly dilated lateral ventricles. Ventriculography a few days later showed very large lateral and third ventricles, and a collection of air in the midline in the superior anterior part of the cerebellum. This was interpreted as a cyst of the cerebellum connecting with the ventricles, and causing occlusion of the aqueduct. It was difficult to explain how the air got into the lateral ventricles when injected into the spinal canal for the encephalogram, unless the obstruction was only partial or unless it was released at times.

A craniotomy was done through a low left occipitoparietal flap, the tentorium was incised and a cyst was exposed and opened. No tumor was found associated with this subtentorial cyst. Following this procedure the patient's temperature became very high and remained elevated until his death a week later. Postmortem examination showed the lateral and third ventricles were greatly dilated. The posterior horn of the left lateral ventricle was herniated through the tentorial notch into the superior aspect of the cerebellum forming a pseudocyst. The aqueduct was completely occluded close to its entrance into the fourth ventricle. Microscopic studies through the midbrain revealed a diffuse proliferation of astrocytes throughout, favoring a diagnosis of astrocytoma, causing occlusion of the aqueduct. Here again the actual primary pathology was in the midbrain and inoperable, but this diagnosis could not be made from the ventriculograms.

Case vii. J. M., white male, aged forty-seven, became ill about four months prior to his admission to the hospital, at which time he developed a very marked diplopia. About one month later he developed a severe frontal headache. Shortly afterwards, he became delirious and was admitted to a local hospital. During his stay in the hospital he seemed very confused. He forgot the names of his children, tried to get out of bed and go home in his hospital clothes, and became very hard to manage. He was very weak at this time and remained at the hospital for ten days, during which time his mental picture gradually cleared up. He became oriented again but his headaches persisted. During the next few months he stayed at home, but continued to complain of a persistent headache and diplopia. About ten days before admission to

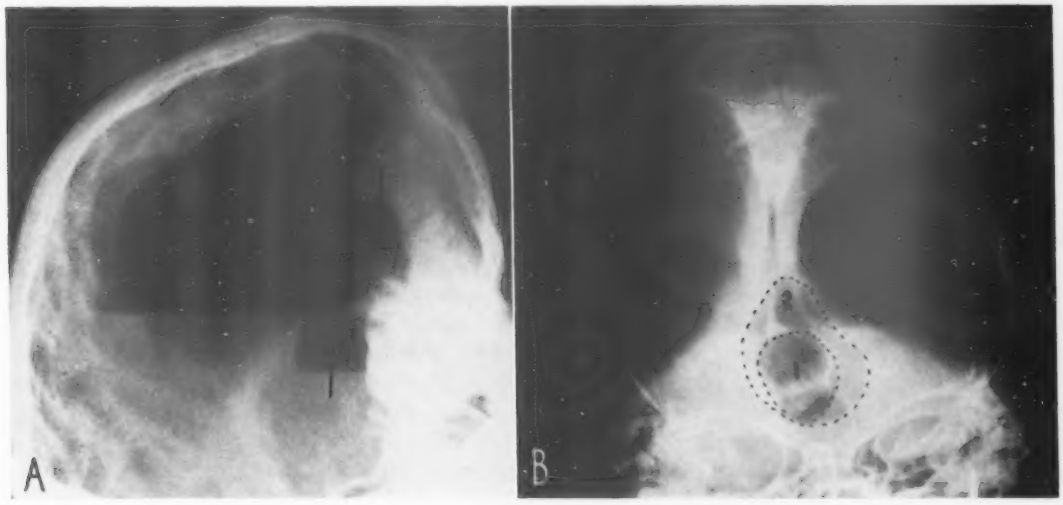

Fig. 6. Case vi. A massive dilatation of the lateral and third ventricles is present. None of the films show any air in the aqueduct. The third ventricle is represented by the collection of gas labelled (*1*). There is another collection of gas present labelled (*2*) which is also outlined by the outer circle of dots in the posteroanterior projection. This was interpreted as a cyst of the cerebellum which communicated with the ventricles. However, it proved to be a part of the left lateral ventricle which had herniated through the tentorial notch into the superior part of the cerebellum. The primary pathology was a diffuse astrocytoma of the midbrain causing occlusion of the aqueduct.

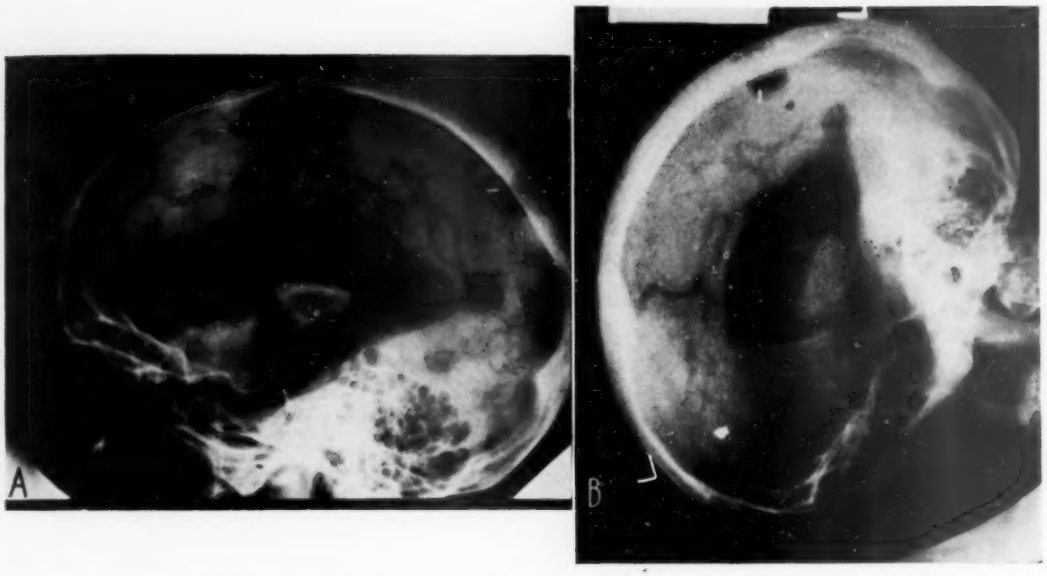

Fig. 7. Case vii. The lateral and third ventricles are dilated. The third ventricle appears to end abruptly as shown by the dotted line a short distance in front of the calcified pineal shadow, *B* (arrow). There is no evidence whatever of any air in the aqueduct or fourth ventricle. A pineal tumor was considered, but surgical exploration of the pineal as well as the postmortem examination showed the pineal gland and the third ventricle to be uninvolved. The aqueduct, however, was occluded beginning a short distance from the third ventricle. The entire midbrain was infiltrated by tumor tissue having the histologic characteristics of a pinealoma. This tumor no doubt arose from an aberrant pineal rest.

the hospital, he again began to show a very poor memory, some involuntary urination and a mild ataxia, tending to fall backwards.

Examination revealed a somewhat disoriented patient, showing definite defects in his sensorium and intellect. There was no elevation of the discs, although the eyegrounds seemed mildly hypertensive. The pupils responded only slowly and incompletely to light, the right pupil being slightly larger than the left. There was an impairment of the upward gaze; the patient never got more than 10° above the horizontal in either eye. The reflexes, both deep and superficial, were equal and normal. The patient had definite difficulty in standing or walking, with a tendency to fall backward and to the right. It was believed at this time that the patient probably had a midline cerebellar tumor, although the early onset of psychotic symptoms and the absence of a choke tended to make one hesitate in making such a localization. The laboratory studies were negative.

Ventriculography (Fig. 7). There is a symmetrical dilatation of the lateral and third ventricles. The suprapineal recess is not visualized and the posterior part of the third ventricle appears to end abruptly about 1 cm. anterior to the calcified pineal shadow. There is no filling of the aqueduct or fourth ventricle. A tumor of the pineal was considered as the most likely diagnosis.

The patient was explored through a high right occipitoparietal flap. The posterior portion of the corpus callosum was separated and the pineal region was exposed and found to be normal. Following the operation the patient's condition was fairly good for about two weeks. He then began to become moribund and died six days later.

Autopsy. Coronal section revealed a complete occlusion of the aqueduct shortly after its departure from the third ventricle. The pineal gland was normal and uninvolved. Microscopic studies showed the entire midbrain to be infiltrated by tumor tissue having the histologic characteristics of a pinealoma. This tumor, no doubt, arose from a pineal rest and by its invasion produced a complete occlusion of the aqueduct.

Comment. In Cases v, vi, and vii the primary pathology was situated around the aqueduct causing obstruction. In none of these cases was it possible to locate the

pathological condition definitely. In each case surgery was done which could have been avoided had we been able to make a correct diagnosis. In Case v, which proved to be a gliosis occluding the aqueduct, we are unable to explain the recurrence of symptoms three months after the third ventricle was opened, particularly in view of the fact that the communication between the third ventricle and the cisterna interpeduncularis was patent at postmortem examination. Herniation of the lateral ventricles through the tentorial notch as occurred in Case vi must be extremely rare. We have no explanation for the apparent obliteration of the posterior part of the third ventricle such as was observed in Case vii.

The third group of cases are still more unusual and extremely discouraging from a roentgenological point of view. In one of these cases the obstruction occurred around the foramina of Luschka and Magendie and still no air was observed within the aqueduct. In the second case no pathological cause could be found at autopsy to account for the dilated ventricular system since no obstruction was present. This case nevertheless presented a typical roentgenological picture of an aqueduct obstruction resembling in most details those air studies already described in the previous cases.

Case viii. S. P., white male, aged forty-six, six months prior to admission suddenly developed severe occipital headache associated with nausea, projectile vomiting, dizziness and blurring of vision. He continued to work on his farm during the next few weeks. He soon began to develop attacks of right-sided weakness and numbness which would last about fifteen to twenty minutes, and would recur many times during the day. These attacks were not associated with any loss of consciousness. His headaches continued and he gradually became more tired and weak and finally was forced to give up his work. About two weeks prior to his admission he suddenly became irritable, impulsive, disoriented, careless of his habits, and extremely difficult to manage. Because of this sudden change in his behavior he was transferred to a local hospital. His headaches and vomiting continued. Shortly before his admission he developed definite convulsive seizures of a generalized type.

Examination revealed the patient to be confused and dehydrated. He had a bilateral choked disc of one diopter. Pupils reacted poorly to light and there was a questionable bilateral abducens weakness. Deep reflexes were absent. The abdominal reflexes were variable but tended to be decreased on the left side. One could discern no significant disturbances of the motor function although cooperation in these tests was very poor. The patient did respond to noxious stimuli. No other sensory examinations were possible because of the patient's inability to cooperate. He would sit about reaching for imaginary objects in space. Regression was very definite and was manifested by his inability to care for his bowel and bladder functions. The clinical impression was that the patient was suffering from a brain tumor, but there were not sufficient clinical findings to indicate the exact location of the tumor. He was therefore transferred to the neurosurgical service where air studies were done.

Ventriculography (Fig. 8). There is a slight enlargement of the lateral and third ventricles, without displacement. In the lateral view with the patient prone and the head resting on the forehead, a small amount of air is seen in what might be the fourth ventricle. This collection of air is not seen in any of the other views. There is no filling of the aqueduct. Some air is present in the cisterna magna. It was difficult to account for this accumulation of air in the cisterna unless one assumed it to have escaped from the fourth ventricle or to have leaked in around the trephine openings at the time the air was injected into the ventricles. We assumed the latter since there was a slight hydrocephalus which indicated some degree of low obstruction and consequently one would not expect the air to pass out readily into the cisterna. Since no localization was possible either clinically or roentgenographically, surgery was not done.

Following the air studies, the patient continued to do very poorly and he died forty-eight hours later. External examination of the brain revealed a diffuse brownish-black tissue that covered the entire base of the brain including the inferior surfaces of the pons, medulla and cerebellum. There appeared to be an obliteration of the foramina of Luschka and Magendie by this tumor. The fourth ventricle and aqueduct were normal. Microscopic sections re-

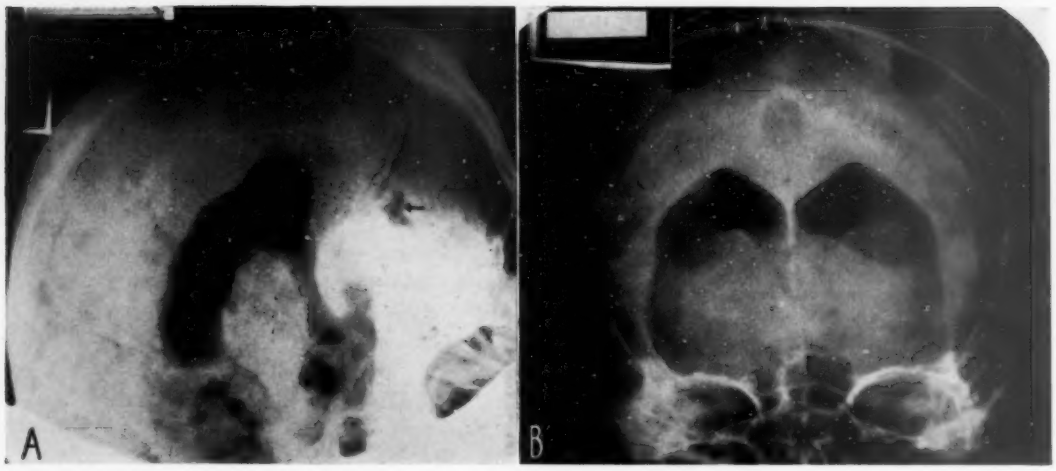

Fig. 8. Case VIII. There is a slight dilatation of the lateral ventricles. The third ventricle is not well demonstrated. There is no sign whatever of air in the aqueduct. In only one view, the lateral with the forehead down, *A*, there is a collection of gas which might be considered as representing the fourth ventricle (arrow). There is a considerable amount of gas in the cisterna magna and some gas in the basilar cisterns and subarachnoid spaces. Postmortem examination of the brain showed a diffuse brownish-black tissue covering the base of the brain including the inferior surfaces of the pons, medulla and cerebellum. The foramina of Luschka and Magendie appeared to be obliterated by the tissue which microscopically was a melanotic meningioma. The aqueduct and fourth ventricle were normal.

vealed this tissue to be a melanotic meningioma, the color being produced by the melanin pigment.

Comment. In view of the postmortem findings in Case VIII it would seem that the correct diagnosis could never be arrived at by ventriculography. Encephalography might have given more information inasmuch as the tumor projected into the basilar cisterns.

Case IX. M. D., male, aged fifty, whose illness dated back about a year and one-half, at which time he complained of malaise, and a visual disturbance, being unable to read for any prolonged period of time. Six months prior to his admission, he developed an attack of vertigo which was so marked that he was unable to get out of his car. He saw a local doctor who told the patient that he was suffering from a hypertension and advised him to rest. Since then he has noted a gradual diminution in his vision until at the time of admission he was practically blind in both eyes. His dizzy spells continued, especially when he drove a car or a team of horses. Two weeks prior to admission he developed a severe tinnitus. He also manifested marked changes in his mood from a person who was very happy and euphoric to one who was very much depressed with a tendency to worry about trivial matters.

Examination revealed a bilateral choked disc of two diopters. The retinal vessels were narrow and tortuous and petechiae were scattered throughout both fundi. The cranial nerves were otherwise intact. The deep reflexes were all hyperactive but appeared to be a little more active on the left than on the right. The rest of the neurological examination was essentially negative. The routine laboratory studies were negative. At the time of examination the differential diagnosis consisted either of a brain tumor or a hypertensive encephalopathy. In view of this difference of opinion, a spinal tap was performed. The fluid was colorless, clear, contained no cells but showed an initial pressure of 28 mm. of mercury. Because of these spinal fluid findings, the diagnosis of a cerebral neoplasm was favored, but there were insufficient clinical findings to accurately localize the lesion.

Ventriculography (Fig. 9). This shows a moderate dilatation of the lateral and third ventricles without any displacement. The upper 1 cm. of the aqueduct is filled with air and appears to be dilated. There is no air in the lower end of the aqueduct or fourth ventricle. These findings were considered as consistent with

either an obstruction about the aqueduct or an obstruction due to a lesion of the cerebellum.

A cerebellar exploration was performed. There was an extensive herniation of cerebellar tissue. No lesion was found. Following the operation the patient's condition gradually became worse. He died four days following the operation.

Autopsy. Gross sections through the brain revealed no pathological lesions to account for the hydrocephalus. There was no obstruction of the aqueduct or fourth ventricle. Microscopic sections revealed a mild diffuse and patchy demyelinization. The midbrain was intact and there were no obstructive lesions in the region of the aqueduct.

Comment. In this case, explanation of the dilated ventricles is extremely difficult unless one assumes an intermittent swelling of the brain with a secondary aqueductal occlusion due to the altered brain tissue.

Until recently tumors of the third ventricle have caused us no great difficulties in localization by ventriculography or encephalography. If no air reaches the third ventricle and the lateral ventricles are dilated and reasonably well filled with gas we have been correct in assuming that the foramina of Monro are blocked or that the entire third ventricle is filled with tumor. If the tumor arises posteriorly and does not fill the ventricle then the outline of the mass has been readily discernible following ventriculography. However, even in one such apparently obvious case a gross error in diagnosis was made.

Case x. N. A., white male, aged twelve, was apparently well until about two weeks prior to admission at which time he suffered a head injury. Since that time he had complained of headaches and a gradual weight loss. A week later he began to vomit and became somewhat lethargic. Examination on admission revealed a limitation of upward gaze. The deep reflexes were reduced in the upper limbs and increased in the lower limbs. There was a generalized muscular weakness. The impression at this time was that the patient probably had a dural hematoma. Four trephine holes in the parietal bones revealed no collections of blood. A few weeks later ventriculography was done.

Ventriculography (Fig. 10). The lateral ventricles were symmetrically dilated but not displaced. A small amount of air was present in the third ventricle. This air was displaced around what appeared to be a tumor mass which arose from the floor and filled the posterior portion of the third ventricle. Because of the size and location of the tumor it was considered inoperable. The patient did not tolerate this procedure very well and died six days later.

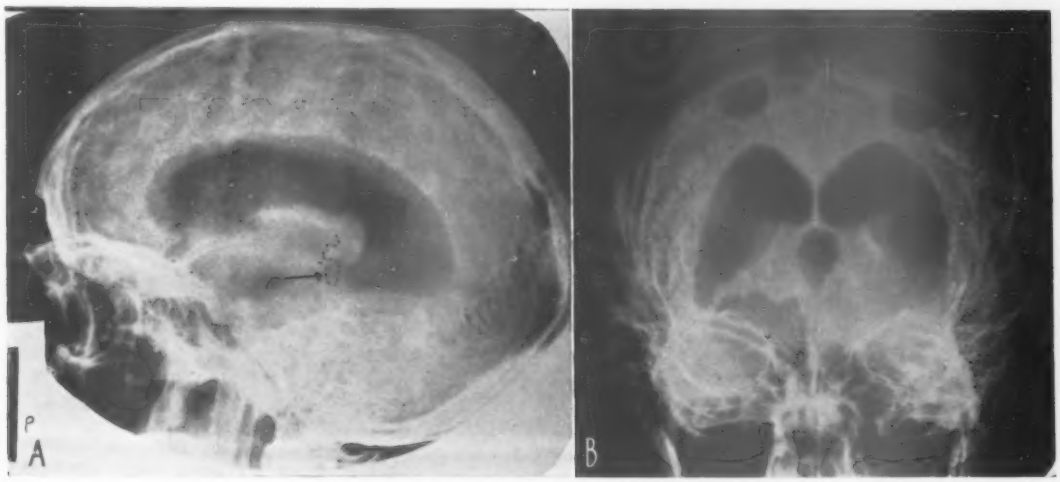

Fig. 9. Case ix. The lateral and third ventricles are dilated and not displaced. A short segment of the upper aqueduct is filled with air (arrow), *A*. There is no gas in the rest of the aqueduct or the fourth ventricle. Postmortem studies showed no tumor or obstruction of any kind in the ventricular system. There was no obvious explanation of the internal hydrocephalus. The aqueduct appeared to be normal even though it was not demonstrated roentgenologically.

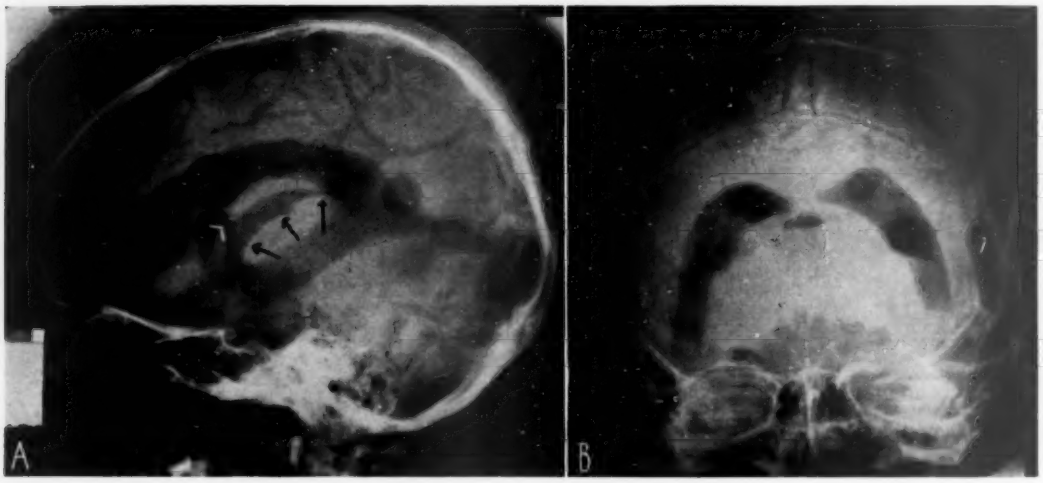

FIG. 10. Case X. The lateral view, *A*, shows what was considered to be a mass (arrows) arising from the floor of the posterior part of the third ventricle. Bony defects in the calvarium and hemostasis clips are the result of an earlier exploration in search for a dural hematoma. The posteroanterior view, *B*, shows the dilated lateral and third ventricles and the upper margin of the supposed mass in the third ventricle. At necropsy a large cerebellar tumor was found which had pushed the midbrain forward and upward. The apparent mass in the third ventricle (arrows) was not a tumor but merely a marked upward displacement of the floor of the ventricle caused by the displaced midbrain.

Autopsy. There was a large cerebellar tumor involving primarily the superior portion of the cerebellum. The tumor mass had extended anteriorly to involve the midbrain. The aqueduct was occluded by the pressure of the tumor but not by invasion. The enlarged cerebellum had been displaced upward displacing the midbrain forward and upward producing a marked elevation of the floor of the posterior portion of the third ventricle. There was no tumor in the third ventricle.

Comment. The roentgen picture produced by this cerebellar tumor was identical with others resulting from lesions within the third ventricle. The air studies alone led to an entirely erroneous localization. Even the clinical findings did not aid in the localization. Such a case is most baffling from the standpoint of accurate localization and subsequent surgical treatment.

Because of the influence which technical factors may have on the demonstration of the various portions of the ventricular system it may be well to describe the technique used in the cases reported above. In the usual case a total of ten films is made. Two upright views are taken first, one anteroposterior and one lateral. Then, with the patient lying on his back, an anteroposterior and a lateral view are taken without changing the position of the patient between the two exposures. In a similar fashion, with the patient lying on his abdomen, a posteroanterior and lateral view are taken. Finally stereoscopic films are made in the right and left lateral prone positions. In some of these cases stereoscopic lateral views were not taken but instead a single lateral plus an anteroposterior exposure was made again without changing the position of the head between the two. This permits one to view films taken at right angles without having any change occur in the distribution of the gas in the ventricles. Our impression now is that neither the stereoscopic laterals nor the right angle films have given us much information which we could not obtain from a single lateral view. In every case the wet films are examined immediately after processing and if any additional views are desired they can be taken at once.

There are two procedures which might be of value which we have not employed in

these cases. In the first place we have not used any special maneuvers in an attempt to fill the fourth ventricle or aqueduct. Recently we have tried manipulating the head according to Lysholm's method, but have not been very successful in filling the aqueduct. In the second place we have not used any of the denser contrast media such as thorotrast or lipiodol as a substitute for air or other gases. It is quite likely that lipiodol in the ventricles could be manipulated by roentgenoscopic control into the aqueduct and fourth ventricle and in that manner their patency could be definitely established. We plan to try this procedure the next time we are unable to localize an obstruction in the midbrain as contrasted with the cerebellum.

SUMMARY

There are a significant number of cases of obstructing lesions in the midbrain and cerebellum in which it is difficult or impossible to accurately localize the pathology either by neurological or by roentgenological methods. Ventriculography fails because absence of filling of the aqueduct and fourth ventricle may occur even though these structures are patent. It is possible that better filling might be obtained if the patient's head were manipulated in some manner such as Lysholm describes for filling the fourth ventricle. If this fails one might visualize the aqueduct with lipiodol which has been injected into the lateral ventricles and then manipulated into the aqueduct by aid of roentgenoscopic guidance. However, if the aqueduct and fourth ventricle are not visualized by the ordinary ventriculographic studies it is not possible to state with certainty whether the obstructing process is in the midbrain or the cerebellum.

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CHEST ROENTGENOGRAMS IN POLYCYTHEMIA VERA AND POLYCYTHEMIA SECONDARY TO PULMONARY ARTERIOLOSCLEROSIS*

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THE differential diagnosis of erythremia (primary polycythemia, polycythemia vera) and erythrocytosis (compensatory polycythemia, secondary polycythemia) has been discussed at length in the literature.[1,2,23,24] In certain instances, however, their differentiation continues to remain a difficult problem. The purpose of this preliminary report is to call attention to roentgenographic lung changes which have aided us in identifying these conditions. While our series of cases is limited, polycythemia with pulmonary arteriolosclerosis occurs so rarely that large series cannot be expected. It is hoped that observations by others in larger groups of patients will permit proper evaluation of these pulmonary findings in the future.

Polycythemia is a disease characterized by a decided and persistent increase in the red cells and hemoglobin per unit volume of blood. It exists as two clinical types: (1) polycythemia vera, and (2) secondary polycythemia.

Polycythemia vera is the term applied to the condition in which excessive erythropoiesis appears to be the primary disorder. Its etiology is unknown. The secondary type includes a much larger group of patients in whom polycythemia appears to represent an effort to compensate for some interference with proper oxygenation of the blood (residence at high altitudes, cardiovascular and respiratory abnormalities).

Cyanosis, splenic enlargement, and hyperglobulinism constitute the cardinal clinical symptoms of polycythemia vera. Headache, vertigo, hemorrhage, and disturbed vision are also common. In addition to high red cell and hemoglobin determinations, these patients present an increased blood volume, increased hematocrit readings, and an elevated basal metabolic rate.[23,24] Leukocytosis with a moderate shift to the left and excessive quantities of platelets are the rule. The sedimentation rate, color index, and volume index, however, are decreased.

Of paramount importance in identifying secondary polycythemia is the presence of an adequate explanation for the increased blood count. Clinically, there is practically nothing to assist one in differentiating between the primary and secondary diseases. It is noteworthy that the blood volume and white cell count are not always increased in secondary polycythemia.

The present treatment of polycythemia has as its purpose the reduction of the elevated red cell count and hemoglobin content of the blood. Methods employed to effect these changes include the use of benzol, phenylhydrazine, venesection, and irradiation. Such therapy being contraindicated in compensatory polycythemia, accurate diagnosis is essential if untoward reactions to the decreased blood count are to be avoided. We have observed a fatality following the use of phenylhydrazine in a patient thought to have polycythemia vera but who, at postmortem examination, was found to have had primary pulmonary arteriolosclerosis with secondary polycythemia.

CLINICAL MATERIAL

We have reviewed the chest roentgenograms of 11 patients with polycythemia vera in whom there has been no clinical suggestion of pulmonary angiosclerosis. In addition, we examined the films of 3 patients in whom the diagnosis of pulmonary

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arteriolosclerosis was either proved at autopsy or strongly suggested on clinical grounds.

Case 1. (Reported by Coman.[6]) The patient, a white woman, aged thirty-five, was admitted to the Hospital of the University of Pennsylvania on April 20, 1935, complaining of a pain in the right costovertebral angle of ten years' duration. She had had a sore throat in December, 1934, following which she constantly felt exhausted and dyspneic. Headache, lacrimation, vertigo, and a sharp stabbing pain at the angle of the left scapula were of more recent occurrence.

Except for marked cyanosis of all the mucous membranes and an accentuated second pulmonic heart sound, the physical examination was negative. The spleen was not palpable. Blood pressure was 130/90. The red blood count was 8,120,000; hemoglobin, 124 per cent. The leukocyte count and differential count were normal.

On April 11, 1935, the patient was started on phenylhydrazine, 0.1 gm. three times daily.

Several days later, April 15, 1935, because of electrocardiographic evidence of right axis deviation and other suggestive evidence of pulmonary arterial disease, phenylhydrazine was stopped. After completely reviewing the patient's findings, polycythemia vera seemed the most probable diagnostic possibility, and phenylhydrazine therapy was resumed several days later.

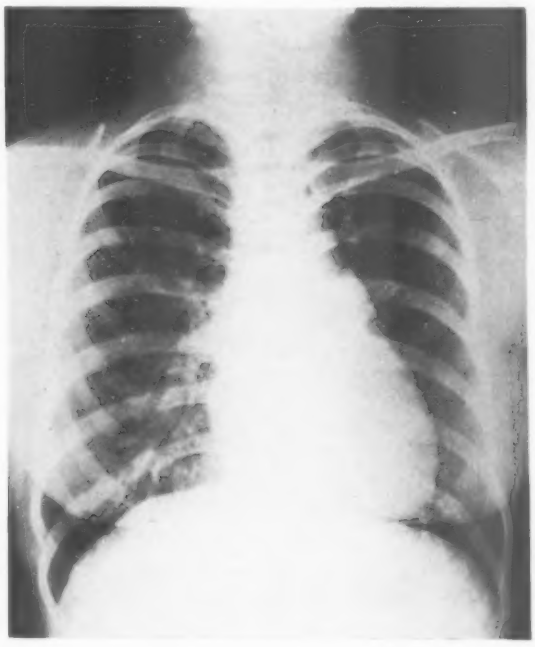


Fig. 1. Case 1. Pulmonary arteriolosclerosis confirmed at postmortem examination. The middle and peripheral zones of the lung are normal. The pulmonary area is prominent.

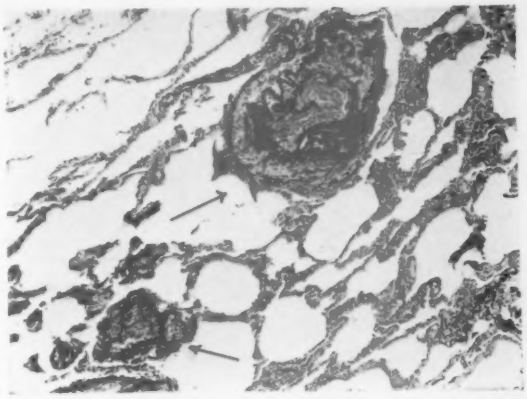


Fig. 2. Case 1. Photomicrograph of a portion of the lung taken from Case 1. Note the complete obliteration of the smaller arterioles due to fibrosis and hyalinization. Stains for elastic tissue showed the internal elastic lamina thickened and spread with a general increase in the elastic elements of the vascular wall.

On April 30, the red blood count was 5,000,000; hemoglobin, 97 per cent. The patient complained of breathlessness and vertigo. Cyanosis became more marked. Paralysis in both lower limbs developed. The patient became stuporous and died suddenly on May 2, 1935.

At autopsy, the right auricle of the heart was greatly dilated and engorged. The aorta was apparently smaller than normal, as were its main branches. Distal to the origin of the left subclavian artery, the aorta presented a constriction which barely permitted the little finger into its lumen. The cut surfaces of the lungs revealed prominent pulmonary arteries which simulated gaping tubes. Their walls were thickened and the larger vessels were dilated.

The spinal cord showed a thrombosis of the posterior spinal artery.

Microscopic examination of the lungs showed complete obliteration of many of the smaller arteries and arterioles by fibrosis and hyalinization (Fig. 2).

Diagnosis: Primary pulmonary sclerosis.

Clinically, cases of this type are frequently mistaken for polycythemia vera, as this one was.

FIG. 3. Case II. Pulmonary arteriolosclerosis confirmed at postmortem examination. The middle and peripheral zones of the lung are normal. The pulmonary area is prominent. (With permission of Drs. F. B. Stephenson and D. K. A. Allen.)

CASE II.* (Reported by Darley and Doan.[8]) A young woman, aged twenty, was first seen because of marked shortness of breath, palpitation, and a constant craving for salt which dated back many years. Physical examination revealed moderate cyanosis. The second pulmonic heart sound was accentuated. Blood pressure was normal. The red cell count was 6,930,000. The electrocardiographic tracing suggested right ventricular preponderance. Roentgenograms of the chest (reported by Drs. F. B. Stephenson and K. D. A. Allen) revealed marked enlargement of the right heart and pulmonary artery (Fig. 3). At postmortem examination the patient was found to have primary pulmonary arteriolosclerosis.

CASE III. The patient, female, aged thirty, was admitted to the hospital complaining of breathlessness and swelling of the legs. The patient's past history suggested congenital heart disease which had been aggravated by recurrent attacks of rheumatic fever. Since early childhood, there had been repeated bouts of decompensation.

Physical examination revealed cyanosis of the mucous membranes and a blood pressure of 96/74. A systolic thrill was felt to the left of the sternum. Red blood cells, 8,700,000; hemoglobin, 125 per cent. Electrocardiographic study revealed right axis deviation.

Roentgenographic study of the chest revealed increased prominence of the pulmonary artery and the right heart (Fig. 4).

Our interest in the use of roentgenograms of the chest as an aid in differentiating polycythemia vera from the compensatory types was activated by observations made by one of us (J. Q. G.)[10] and by others,[3] of the capillaries and subcapillary plexuses in living patients with polycythemia. In general, the capillaries and venules in the patients with polycythemia vera were found distended and engorged, whereas the patients with marked arteriosclerosis plus polycythemia presented a more normal vascular bed (Fig. 5). This information and the knowledge that vascular engorgement was the most striking anatomic feature of polycythemia vera prompted us to review the chest roentgenograms of the patients reported above for evidences of increased prominence of the vascular markings. The films revealed no roentgen evidence of

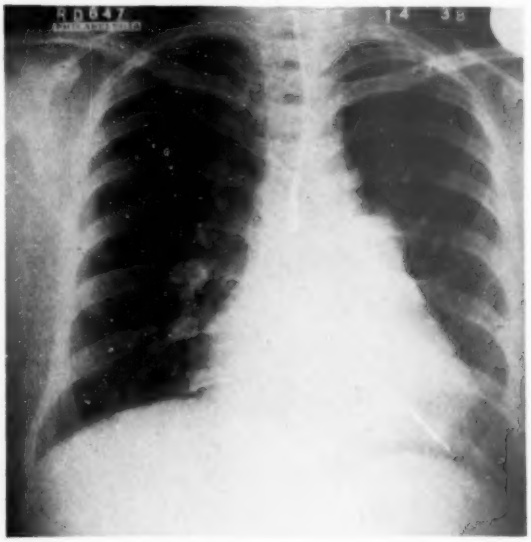


FIG. 4. Case III. Pulmonary arteriosclerosis. The patient is still living. The appearance of the lung fields simulates that seen in Figures 1 and 3. Note the marked increased prominence of the pulmonary area.

* We are indebted to the authors for their kindness in allowing us to include this case in our report.

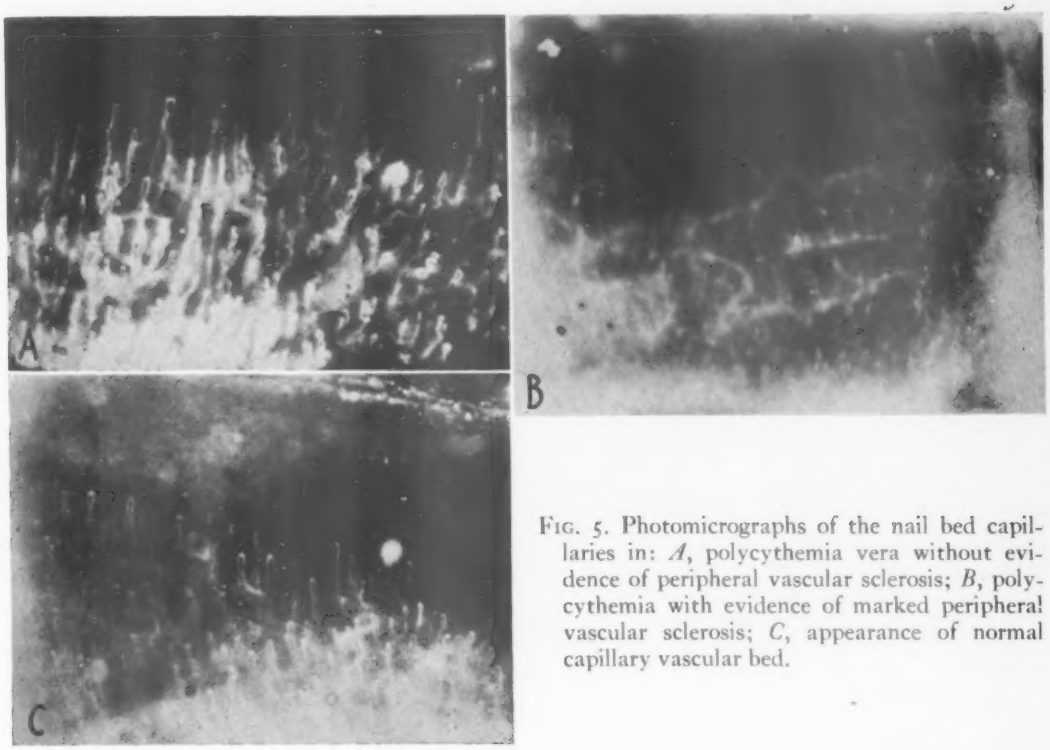


Fig. 5. Photomicrographs of the nail bed capillaries in: *A*, polycythemia vera without evidence of peripheral vascular sclerosis; *B*, polycythemia with evidence of marked peripheral vascular sclerosis; *C*, appearance of normal capillary vascular bed.

vascular engorgement (Figs. 1, 3 and 4).

Roentgen Appearance of the Chest in Polycythemia Vera. That patients with polycythemia vera present marked increased prominence of the truncal shadows of the lungs (Figs. 6 and 7) has long been recognized by those interested in the chest. Pancoast and Pendergrass[18,19] have repeatedly called attention to this in their considerations of the pitfalls in roentgen diagnosis of early silicosis. In our experience, these abnormal vascular markings have been present only in patients with clinical evidence of cyanosis and vascular engorgement. Patients with polycythemia vera examined during a remission failed to reveal similar vascular prominence. It must be remembered that with the advent of middle life, the lung markings normally become prominent,[20] continuing to increase in prominence with age. The evidence seems to indicate that the exaggerated truncal shadows seen during exacerbations of the disease are roentgen manifestations of marked pulmonary vascular engorgement. That such vascular engorgement exists has been amply demonstrated at the operating table and at postmortem examination.[1,3,24]

The importance of sharply defined posteroanterior stereoscopic films made during late diastole cannot be over-emphasized. In this manner, gross exaggerations of vascular markings and other abnormal densities seen in a single flat film of the chest are avoided and properly evaluated. Oblique examinations and films of varying densities may also be of considerable assistance in delineating these shadows.

Recently, Hirsch[11] called attention to the presence of discrete pulmonary lesions in patients with polycythemia vera which we have also observed in three patients. Occurring in the middle zone of the lung (Figs. 7 and 8), these lesions are spherical, sharply circumscribed, and not surrounded by evidences of pulmonary infiltration. According to Hirsch, the lesions developed and regressed rapidly, all in a period of approximately three weeks. In our experience, these nodulations have remained unchanged for years and have been seen only in patients with polycythemia vera.

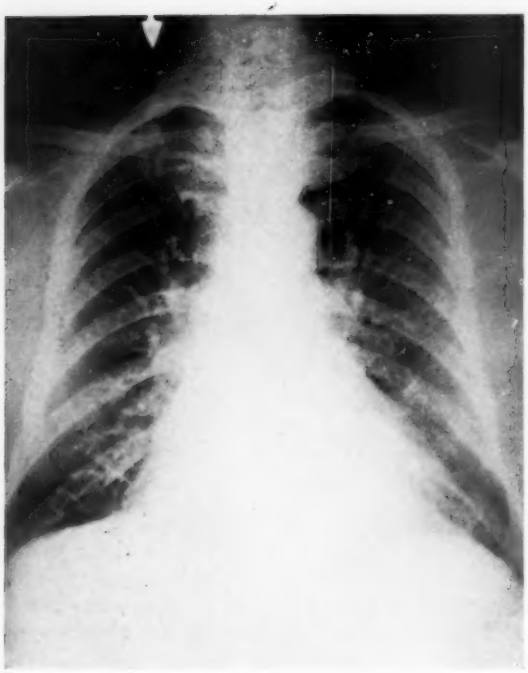


Fig. 6. Polycythemia vera confirmed by postmortem examination. The middle and peripheral zones of the lung reveal increased prominence of the vascular markings.

The morphology of these lesions is unknown. Rodes[21] reported the autopsy findings in a patient with polycythemia vera and pulmonary lesions not unlike (roentgenographically) those reported by Hirsch

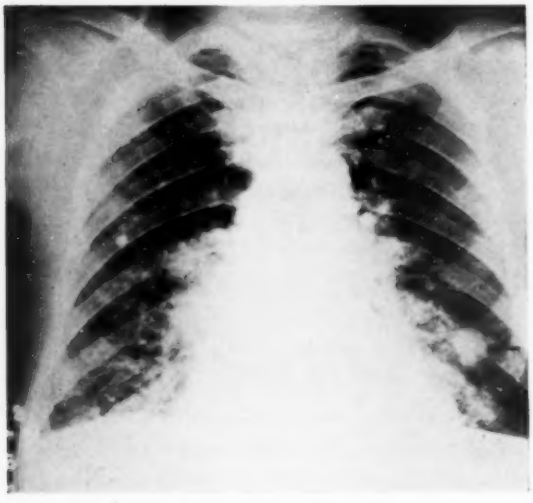


Fig. 7. (We are indebted to Dr. Herman W. Ostrum of the Philadelphia General Hospital for this illustration.) Polycythemia vera confirmed by postmortem examination. All of the pulmonary markings are considerably increased in prominence. Note the nodule in the left lower lung field.

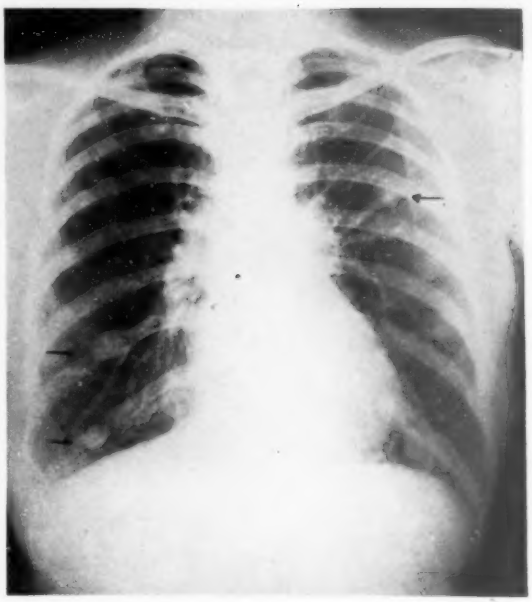
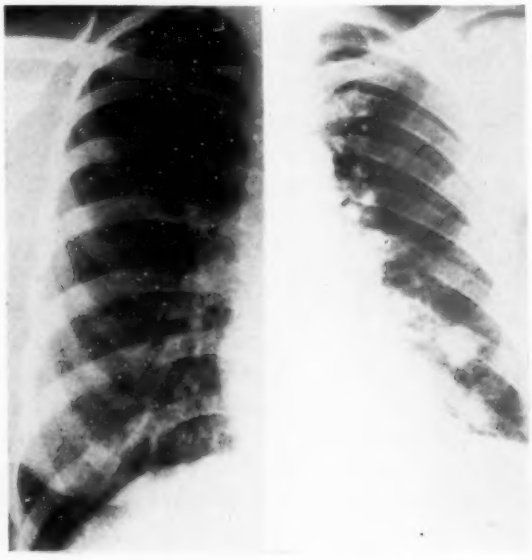


Fig. 8. Polycythemia vera. Blood count normal at the time this roentgenogram was made. Note the nodules in both lower lung fields.



A B

Fig. 9. *A*, appearance of lung field in patient with polycythemia secondary to pulmonary arteriosclerosis. *B*, appearance of lung field in patient with polycythemia vera.

Fig. 10. Diagrammatic representation of pulmonary and vascular changes in: *A*, polycythemia vera; *B*, polycythemia secondary to pulmonary arteriosclerosis. In *A*, note the marked increase in the prominence of the vascular shadows which extends to the periphery of the lung. Note the nodule in the left lower lung field. The cardiac silhouette is essentially normal. In *B*, the mid and peripheral zones of the lung do not present marked engorgement of the vascular shadows. The pulmonary area and silhouette of the right heart are increased in prominence.

which proved to be cavernous hemangiomas. The evanescence of the lung nodules Hirsch described seems to militate against considering all similar lesions in patients with polycythemia vera to be hemangiomas.

The predilection of patients with primary polycythemia to infarction suggests a relationship between this tendency and the pulmonary lesions. The spherical character of the nodules makes one hesitate to accept this as an explanation since pulmonary infarcts are usually wedge-shaped, the apex pointing toward the hilum where the vessels enter. In addition, the usual history of pulmonary infarction is lacking.

According to Krumbhaar[13] and Custer,[7] these lesions may be due to thrombosis in the pulmonary veins, though postmortem studies are lacking to confirm their belief. Stasis infarction plus diapedesis or a rupture in the vein proximal to a thrombus with perivascular hemorrhage seem to explain the clinical observations more adequately. Such extravasations would account for the spherical characteristics of the nodules and the absence of clinical signs of infarction. Once developed, the subsequent course of the lesion would depend upon the degree of resorption or replacement fibrosis occurring in the diseased area.

Roentgen Appearance of the Chest in Secondary Polycythemia Due to Pulmonary Arteriosclerosis. Our experiences with chest roentgenograms in secondary polycythemia have been limited to examinations made of patients with pulmonary vascular sclerosis. In these instances, the middle and peripheral lung fields appeared normal provided pulmonary congestion due to cardiac failure had not developed (Figs. 9 and 10). Abnormally prominent vascular markings and spherical nodules of the type seen roentgenographically in erythremia were not observed in these cases. A brief review of the literature[5,9,12,15,16,17,22] seemed to substantiate these observations. The evidence suggests that this absence of abnormal vascular markings may be due to the proliferative disease processes going on in the pulmonary arteries which narrows them (Fig. 10), thereby decreasing the quantity of blood flowing through their lumina and

into the veins. However, roentgen changes in the hila and cardiac silhouette have been observed frequently in patients with pulmonary arteriosclerosis and secondary polycythemia.

Usually, in the latter condition, films of the chest reveal considerable increased prominence of the hila and pulmonary conus (best seen in the right oblique position)[4] plus dilatation and hypertrophy of the right heart. These are the compensatory changes which follow obstruction and hypertension in the lesser circulation of the lungs in pulmonary arteriosclerosis.

SUMMARY

Polycythemia vera and polycythemia due to pulmonary arteriosclerosis have many common clinical and laboratory findings. Since proper treatment depends upon accurate diagnosis, all available diagnostic procedures should be employed to reach the proper conclusions. Roentgenograms of the chests of individuals with these conditions have revealed pulmonary changes which may prove to be of differential diagnostic value.

Of 11 patients with polycythemia vera, 8 presented abnormally prominent vascular markings roentgenographically. These changes were not observed in the patients with polycythemia secondary to pulmonary sclerosis (Figs. 9 and 10). Instead, the latter group revealed unusual prominence of the right heart and pulmonary conus, the middle and peripheral zones of the lung, being essentially normal.

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LOCALIZED SUBACUTE PHLEGMONOUS GASTRITIS (SO-CALLED BOTRYOMYCOSIS) SIMULATING PYLORIC CARCINOMA

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PRIMARY phlegmonous gastritis is a rare occurrence. Eliason and Wright in 1938 mention, aside from their 31 cases, 276 cases previously described by others. Since then, Lyall has reported 7 cases and Barnett and Harris have reported 1 case. Botryomycosis of the stomach has not been reported so far, although the following 4 cases involving other abdominal organs have been reported: 1 by Berger, Vallée and Vézina (bartholinitis), 1 by Plaut (granuloma of the abdominal wall) and 2 by Kimmelstiel and Oden (granulomas of the intestinal wall, caused by impacted fish bones).

Botryomycosis is a misleading term used to designate a subacute inflammatory process resulting in the formation of granulation tissue with abscesses, in which masses somewhat resembling actinomycosis but actually composed of coalescent tissue debris and staphylococci are found. This condition was at first observed in animals and thought to be due to a distinct species of staphylococcus, which was then called botryococcus. Magrou, in 1914, however, proved that the cocci found in the lesions were merely staphylococci of various strains.

Lately Kimmelstiel and Easley have succeeded in the experimental production of the lesion in rabbits by the introduction of fish bones infected with staphylococci into the intestinal wall and have concluded that botryomycosis is due to a continuous invasion of the tissues by small numbers of staphylococci, foreign bodies perpetuating the infection.

Botryomycosis, as such, is not mentioned in the radiological literature so far as we could ascertain. Nonspecific acute, chronic, diffuse and localized gastritis, however, are mentioned. Haudek (quoted by Feldman) believes that the ulcerative, localized type roentgenologically can easily be confused with carcinoma but that the presence of mucosal folds, occurring in these cases in the constricted area, facilitates the correct roentgenological diagnosis.

FIG. 1. Stomach at the first examination. Note incisura between antrum and pylorus and narrowing of the pyloric canal.

Olsson in his description of the roentgenological features of 2 cases also emphasizes the demonstrability of the mucous coat in spite of the marked thickening of the rest of the wall of the stomach.

In view of the scarcity of information pertaining to this peculiar pathological entity, we believe the following case is of sufficient interest to be reported.

FIG. 2. Stomach at second examination. Note complete annular filling defect.

FIG. 3. Stomach at the end of six hours. Note residue in stomach and small amount of barium in small bowel.

CASE REPORT

The patient, a white housewife, aged fifty-nine, before her marriage a nurse and now a druggist's wife, was about ten years ago subjected to an appendectomy, before and after which, until March, 1934, she was apparently healthy. In March, 1934, she began to suffer from distress in the upper abdomen which became progressively worse. Roentgenological examination, by others, at that time disclosed a non-functioning gallbladder. The clinical diagnosis of cholecystitis was made. The patient was operated upon on January 2, 1935. Numerous soft adhesions were found between the duodenum, pylorus and head of the pancreas. Because of profuse bleeding from these adhesions cholecystotomy was performed. The patient after a precarious convalescence lasting thirty-two days was discharged and remained healthy until October, 1939. At this time she

FIG. 4. Resected pyloric portion of stomach. Note thickening of wall with large abscess and obliteration of lumen.

suffered a fall, shortly after which she began to complain of epigastric pain, distress after meals and intermittent nausea but no vomiting.

Physical findings at this time were essentially negative except for tenderness and the presence of muscular spasm in the right epigastrium.

Ambulatory roentgen examination was performed on January 23, 1940. The esophagus, the cardia and the antrum appeared to be normal. The mucosal folds were of normal size and arrangement. The peristaltic waves were normal to a point about 2 inches from the py-

lorus. At this point the peristalsis stopped and a deep incisura was seen. The lumen of the pylorus showed the mucosal folds to be faintly discernible and of a normal pattern. The stomach emptied normally. The opinion was, at this time, that there existed definite changes in the pylorus, the identity of which could not be definitely established, that resembled a new growth. It was decided to send the patient home and repeat physical and roentgenological examinations later.

The patient was admitted to the hospital on February 27, 1940. She then complained of almost constant nausea but no vomiting. The distress in the upper abdomen persisted and she had lost some weight. A definite mass could be palpated about one inch above the umbilicus.

Laboratory Findings, February 27, 1940. Blood: red blood cells, 3,650,000; white blood cells, 10,400; hemoglobin, 69 per cent; differential: eosinophiles, 15 per cent; stab cells, 2 per cent; segmented, 54 per cent; lymphocytes, 29 per cent. Urinalysis was essentially negative; sediment contained a few leukocytes and epithelial cells with occasional hyaline and granular casts.

Fig. 5. Incompletely developed abscess in the submucosa with so-called botryomycotic granules (low power).

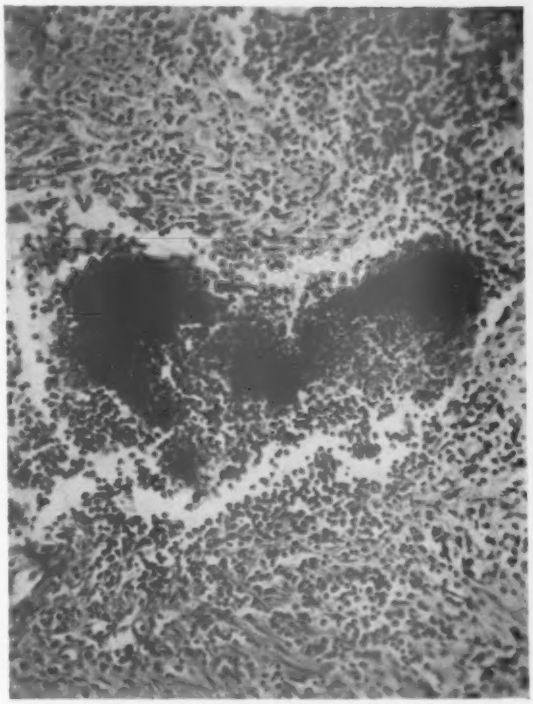

Fig. 6. Fully developed abscess in the submucosa with so-called botryomycotic granules (high power).

Roentgenological examination on February 27, 1940, revealed an almost complete obstruction in the region of the pylorus with hyperperistalsis of the antrum adjacent to the obstruction. Relief examination of the pylorus revealed no evidence of mucosal folds nor ulceration. At the end of six hours there was 90 per cent gastric residue, with a small amount of barium in the small intestine. Notwithstanding the fact that this lesion had grown very rapidly, the diagnosis of an annular carcinoma of the pylorus was made.

Roentgenological examination of the gallbladder at this time revealed the organ to fill and empty normally. No negative or positive shadows could be demonstrated.

The patient was operated upon on March 2, 1940 (by Dr. B. W. Hole). A partial gastrectomy with posterior gastroenteroanastomosis was performed. On the third day following operation, the patient's temperature rose (to 109° F. shortly before death) and she died. Permission for an autopsy could not be obtained.

Pathological Examination. The resected portion of the stomach measured 6 cm. in length and from 3.5 to 4 cm. in diameter. The serosa

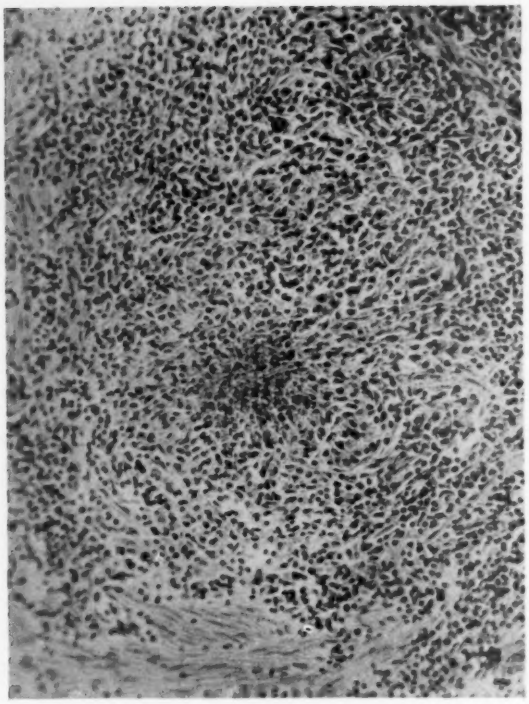

FIG. 7. Tubercle-like formation with central necrosis and epithelioid cells.

covering the major curvature was diffusely somewhat thickened but smooth. Elsewhere, the serosa, which was of a grayish-red color, was covered with fairly firm, quite elastic, pinkish-gray adhesion strands. The wall of the stomach was of a quite firm but somewhat doughy consistency. On section the lumen was almost completely obliterated by the thickening of the wall. The wall measured, in the proximal portion, 14 mm. and in the distal portion 22 mm. in thickness. The increased thickness was caused mostly by a very marked swelling of the submucosa, which measured from 10 to 16 mm. in thickness. There were numerous, quite well circumscribed, soft, pale yellow abscesses, the largest of which measured about 12 mm. in diameter. The subserosa and muscularis showed marked edema. The mucosa covering the lesser curvature measured from 2 to 6 mm. in thickness, its surface being red and uniformly granular. No mucosal folds could be seen. Along the larger curvature of the proximal portion the mucosa was not perceptibly thickened.

Eight blocks of tissue were taken for examination from all layers of the stomach. Hematoxylin-eosin, Gram stain and Ziehl-Neelsen stains were used.

Microscopic Examination. The mucosa was quite well preserved throughout and showed polypoid proliferation at the proximal margin of the lesion. No signs of malignancy were present. The mucosa showed edema and the presence of numerous eosinophile leukocytes, plasma cells and lymphocytes mainly in the interstitium. The muscularis mucosae, submucosa, muscularis and subserosa showed very marked edema. There were also large numbers of inflammatory cells, consisting of polymorphonuclear leukocytes, many of which were eosinophiles, plasma cells and lymphocytes. These were in part diffusely distributed but many of them were found in abscesses, equally present in the muscularis mucosae, submucosa and to a lesser degree in the muscularis. The abscesses varied in size and were partly surrounded by a zone of necrosis of varying width. Within the abscesses were found partly pink and partly blue staining (hematoxylin-eosin stain) masses, which at the first glance appeared to be composed of central fibers and peripheric clubs, resembling actinomyces. However, closer examination revealed that the fibers and clubs were in reality more or less necrotic connective tissue fibers and coalescent necrotic nuclei intermingled at the periphery with masses of rather unevenly staining staphylococci (Gram stain). Sections from the proximal margin of the lesion showed tubercle-like formations consisting of epithelioid cells in radial arrangement surrounding small central areas of coagulation necrosis. These in turn were surrounded by polymorphonuclear leukocytes and plasma cells. There were no giant cells. No acid-fast bacteria were found. The small blood vessels in the muscularis and subserosa showed intimal prolif-

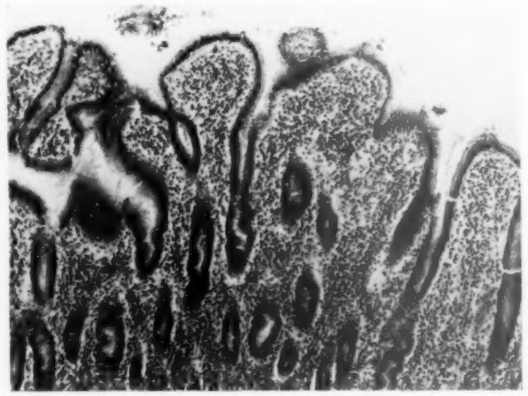

FIG. 8. Hypertrophic gastritis at the proximal margin of the lesion.

eration and some old thromboses. No cultures from the lesions were attempted, as the correct diagnosis was made only after the specimen had been fixed.

COMMENT

The case described presented, aside from the rather nondescript clinical history, decidedly unusual roentgenological and pathological features. Within a period of one month the pyloric end of the stomach, which at first showed a moderate and fairly well localized rigidity of the wall without any filling defects or gross irregularity, had presented the findings of very marked thickening of the wall and almost complete occlusion of the lumen. The mucosal pattern was faintly visible during the first examination and was completely obliterated at the time of the second examination. This is a direct contradiction to the criteria which according to Haudek and Olsson are characteristic for the roentgenological diagnosis of phlegmonous gastritis. The mucosal pattern could apparently not be demonstrated in this case for two reasons: (1) the affected portion of the stomach did not fill at the time of the second examination; (2) the mucosal folds were obliterated by the swelling of the wall. Therefore, the only possible roentgenological diagnosis was annular carcinoma of the pylorus. Even now, after due consideration of all factors, it is impossible to point out any significant features leading to a correct differential diagnosis.

Anatomically the lesion closely resembles those described by Berger, Vallée and Vézina, Plaut, and Kimmelstiel and Oden. No distinct cuticula or clubs could be demonstrated in this case. The granules found in the abscesses consisted of coalescent reticulum fibers, cell nuclei and, in the periphery, of unevenly staining staphylococci. The tubercle-like formations consisting of epithelioid cells surrounding small areas of necrosis, seen in this case, in our opinion, signify the earlier phase of the inflammatory process. No foreign body could be demonstrated anywhere in the wall of the stomach. On the other hand, chronic hypertrophic gastritis was present and could presumably be the primary lesion. In the absence of an autopsy it is impossible to express a definite opinion on the mode of entrance of staphylococci into the tissue. In this instance it is true, nevertheless, that the phlegmonous gastritis was a localized process and therefore likely to be the consequence of entrance of the causative organism from the mucosal surface into the tissue.

SUMMARY AND CONCLUSION

A case of primary localized phlegmonous gastritis resembling so-called botryomycosis in a white woman, aged fifty-four, is reported. The lesion roentgenologically could not be differentiated from an annular carcinoma of the pylorus. Histologically the lesion consisted of granulation tissue with abscesses in which so-called botryomycotic granules, consisting of coalescent tissue and staphylococci, were found. Chronic hypertrophic gastritis is believed to be the cause of the lesion.

We greatly appreciate the permission of Dr. B. W. Hole to investigate this case.

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PANCREATIC LITHIASIS

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PANCREATIC lithiasis is a rare condition. Opie found 2 cases in 1,500 postmortem examinations and Barron 3 cases in 3,457. In 1935 Bost, reviewing the literature, found 79 cases of pancreatic lithiasis reported at autopsy and 28 cases at operation. The latter included one patient whom he had operated upon twice for pancreatic lithiasis. There was roentgenological evidence of single or multiple stones in only 3 of the 28 operated cases. In 2 of these cases the preoperative roentgenological diagnosis of calculi was not made, but evidences of calculi were found in reviewing the roentgenograms after operation. Recently, Engelstad and Römcke reported 2 cases, and Lüdin and Hoechstetter each reported one case, diagnosed roentgenographically. Gillies also reported one interesting case of multiple facetted pancreatic stones, and gave an exhaustive survey of the literature on this subject.

During the five months following April 1, 1938, we have observed 3 cases of pancreatic lithiasis in the Department of Roentgenology of the New York Polyclinic Medical School and Hospital among approximately 240 roentgen examinations, which included the pancreatic area. We wish to present these cases because of their rarity and their importance from the roentgenological and clinical viewpoints.

REPORT OF CASES

CASE I. S. W., male, white, aged forty-five, actor by profession, suffered from epigastric distress after meals, but he has never had severe attacks of pain. Radiating pain and backache were absent. Two years ago he underwent a cholecystectomy. The operative findings could not be ascertained. His complaints persisted and he was referred to the X-Ray Department for roentgen examination of the gastrointestinal tract. This examination was essentially negative, with the exception of numerous foci of increased density scattered throughout an area corresponding closely to the location, size and shape of the pancreas (Fig. 1). These foci were discrete with a tendency to be rounded or oval in shape and averaged 2–3 mm. in diameter. Some of the foci were somewhat larger and more

FIG. 1. Case I. Numerous small calculi scattered throughout the pancreatic ducts.

irregular. Their distribution suggested that they were located in the pancreatic ducts. The involved area was seen extending from the region of the duodenal loop, below the pylorus, upward and to the left where it was seen above and adjacent to the lesser curvature of the stomach and then transversely to the left upper quadrant of the abdomen to the hilum of the spleen (Fig. 2).

CASE II. T. P., male, white, aged thirty-three, salesman by occupation, complained of attacks of sharp, boring pain in the epigastrium during the month of December, 1932. The pain was continuous with slight variation in intensity lasting six days. In February, 1933, he was admitted to the hospital following a recurrent attack of pain and was operated upon after a five day observation period. The operation, done by Dr. F. S. Bancroft, revealed a stone in the ampulla of Vater and a moderate degree of cholecystitis. The head of the pancreas was

twice its normal size, very firm and very nodular throughout. This was considered to be due to chronic pancreatic edema, secondary to cholecystitis. The gallbladder was removed. The patient had a rather uneventful recovery. One year after the operation the pain reappeared and recurred at intervals several times each year, each attack lasting fifteen minutes to four days. During the attacks, the patient was nauseated and sometimes vomited. Fever and jaundice were absent. The last attack of acute pain occurred nine months before our roentgen examination. At this time the patient stated that he had had a dull pain in the upper abdomen at irregular intervals. The pain had no connection with the taking of food and had been present for the previous three weeks. During this time the patient had never been nauseated and had an excellent appetite.* The roentgen examination revealed slight enlargement and slight irregularity of the rugae, characteristic of gastritis. The duodenal cap presented irritability without evidence of an ulcer. The proximal part of the second portion of the duodenum was narrowed moderately by

Fig. 2. Case I. The relative position of the pancreatic calculi to the stomach and duodenum is shown.

* We are indebed to Dr. Samuel Weiss for the clinical data.

Fig. 3. Case II. The findings are essentially the the same as shown in Figure 1.

periduodenal adhesions, which probably followed the cholecystectomy. In the pancreatic area there are foci of increased density of the same distribution as in Case I (Fig. 3). Plain films of the abdomen were made at intervals during the five months following the first examination. Each film confirmed the first findings and showed no change in the distribution or general appearances of the calculi.

The laboratory findings were as follows: The test meal showed normal acidity. The blood sugar 136 mg. per 100 cc. The bile showed normal activity of trypsin and steapsin, and decreased activity of amylase. There was no glucose in the urine.

Case III. M. M., housewife, white, aged fifty-two. This patient was operated upon one year before our roentgen examination. Appendix and gallbladder were removed with complete relief of the symptoms up to the present time. During the last four to five weeks she has experienced rather constant, and at times, severe abdominal pains, located chiefly in the epigastric region and in the left upper quadrant of the abdomen. Radiation of the pain was absent. The attacks of pain had no relation to the

Fig. 4. Case III. Calculi lodged in the head and tail of the pancreas.

taking of food. Jaundice and fever were never noticed. Nausea occasionally was present during the attack.*

The roentgen examination revealed irritability and slight irregularity in outline of the first portion of the duodenum. Two discrete areas of increased density were noted in the region of the head of the pancreas, and another in the region of the tail. The distribution and general appearances were essentially the same as in Cases I and II. No foci were noted in the region of the mid portion of the pancreas (Fig. 4). The tests of the duodenal contents for ferments and glucose tolerance revealed normal values. There was no glucose in the urine.

DISCUSSION

We believe that in all 3 cases we are dealing with multiple stones in the pancreatic ducts; thus far, we have been unable to prove this, either surgically or by postmortem examination. However, the relative position of the involved area to the stomach and duodeum, as well as its shape, supports the assumption that the calcified foci are located in the pancreas. Borst, Lindsay and Wolf-Tietze reported cases showing similar roentgen findings, which proved to be multiple stones in the pancreatic ducts. Römcke and Schoendube also found stones in the pancreatic ducts at postmortem in cases presenting similar findings.

From the foregoing it is evident that pancreatic lithiasis is infrequently diagnosed, the reasons for which are (1) that the condition is rare, as the statistics show; (2) during the rush of routine the plain films of the abdomen are often omitted from the gastrointestinal examination, and calcification may be mistaken for residue of barium in the small intestine or stomach, and (3) that there are several other pathological possibilities which may exist and be erroneously evaluated; for instance, in Case III a few calcified foci were seen adjacent to the lesser curvature of the stomach, and the possibility of a peptic ulcer crater or niche was considered (Fig. 5). After further study, however, the calcified foci were recognized

Fig. 5. Case III. The relative position of a pancreatic calculus to the stomach may simulate an ulcer of the lesser curvature of the stomach.

* We are indebted to Dr. H. Chase for the clinical data.

as pancreatic stones, together with the other calcified foci in the region of the pancreas which were found on the same films, and the same findings were present on the films one week following the barium feeding. In another instance the calcifications were mistaken for calcified lymph nodes in the cases reported by Ackman and Ross and Faust.

In the differentiation of calcifications involving the left hypochondrium the normally high position of the tail of the pancreas in the upper left abdominal quadrant should be kept in mind. This high position of the tail of the pancreas on the roentgenogram is a fact which is at variance with the general concept of its location. In the cases reported in this paper the calcification in Case I appeared in the projection of the calcified costal cartilage of the tenth and eleventh ribs, in the projection of the twelfth rib in Case II, and at the level of the tenth and eleventh dorsal vertebrae in Case III. Pancreatic stones might therefore be mistaken for calcification in a costal cartilage. A lateral view or stereoscopic films would aid in this differential diagnosis.

None of our patients had the colicky pain in the left hypochondrium with radiation to the left shoulder which is considered characteristic of pancreatic lithiasis. However, all 3 patients had dull epigastric pain and tenderness, and in addition, the second case complained of occasional severe attacks of boring pain in the epigastrium, while the third had attacks of violent pain in the left hypochondrium. The attacks of pain were not associated with the taking of food. Some of the 28 operated cases of pancreatic lithiasis reported by Bost had no pain at all, others had dull soreness or cramps, others violent attacks of pain in the epigastrium, with or without radiation to the lumbar region. These histories seem to prove that epigastric pain is a vague and unreliable symptom and when present it is necessary to think of pancreatic lithiasis, as well as of other pathological conditions in this area, even when the pain does not radiate to the left shoulder. One patient of Bost had radiating pain, but this was atypical in distribution, as it extended along the dorsal spine in the interscapular area. In another case the pain radiated from the left upper quadrant to the iliac fossa. Engelstad and Römcke reported a similar case. These latter cases show us that in the differential diagnosis of pancreatic lithiasis the diseases of the genitourinary tract must also be considered.

Laboratory findings also help in the recognition of pancreatic lithiasis. The blood sugar in Case II was somewhat increased, showing 136 mg. per 100 cc. The glucose tolerance test was negative in Case III. These findings conform with those of the cases surveyed by Bost. Glycosuria was present in 7 of his 28 cases. Oser reported glycosuria in 24 of 70 cases. The duodenal contents had a decreased amylase activity in our Case II, and was normal in Case III. Unfortunately no laboratory examinations were made in our Case I.

In Case II, when operated upon for violent epigastric pain, a stone was found impacted in the ampulla of Vater. The pancreas was found to be increased in size, nodular and firm, indicating a chronic pancreatitis. Following the cholecystectomy the same type of severe attacks of pain recurred. The other two patients also experienced no improvement following cholecystectomy. In the 28 cases surveyed by Bost, gallstones were found in only three. These observations render it unlikely that pancreatic lithiasis is always secondary to gallbladder disease. Moreover, a gastroduodenitis spreading to the pancreatic ducts and causing obstruction, or obstruction of the pancreatic ducts, due to a tumor or stone with secondary infection, might cause pancreatic lithiasis. Experimentally, in dogs, Lazarus succeeded in producing a soft concretion the size of a pinhead by infecting the pancreatic ducts with colon bacilli and subsequently ligating a portion of the pancreas, producing a small cyst in which the concretion developed and was found forty days after the infection. Two other dogs, similarly treated, developed an acute sup-

purative pancreatitis and died. The combination of infection and ligation of the ducts was apparently necessary to produce concretions, as in 6 cases, ligation alone, and in 3 cases, infecting the ducts alone, failed to produce concretions.

We have not had the opportunity to inspect and analyze the concretions in our cases, but analysis of pancreatic concretions made by other observers found them to be composed of calcium phosphate, or calcium carbonate and organic substances.

As to therapy, we wish to mention that single and multiple stones have been removed from the pancreas successfully, but it is doubtful whether surgery would be successful in cases similar to ours because of the great number of the stones and their scattered distribution.

SUMMARY

Three cases of pancreatic lithiasis diagnosed roentgenologically are described.

The high position of the tail of the pancreas in the left hypochondrium should be borne in mind.

Lateral views or stereoscopic films are important in the differential diagnosis of pancreatic stones and other calcifications.

The location and character of the pain in pancreatic lithiasis are not characteristic in many instances.

Glucose tolerance test and ferment determination of the duodenal contents might be of diagnostic value in these cases.

Infection and simultaneous obstruction of the pancreatic ducts are considered to be the etiological factors in pancreatic lithiasis.

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TUMORS IN ONE OF HOMOLOGOUS TWINS*

HODGKIN'S DISEASE; OSTEOGENIC SARCOMA

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WERE sufficient clinical material available, the study of homologous twins would seemingly contribute a great deal toward solving the genetic theory of cancer. As it is believed that both individuals of such twins arise from the same impregnated ovum, if one has cancer, the other should of necessity have cancer too. McFarland and Meade[1] report such an occurrence in a number of cases in certain types of tumors, including carcinoma of the breast and Hodgkin's disease. As the authors express it, "tumors occurring in homologous twins seem always to be similar, symmetrical and simultaneous."

That this rule does not always hold true was shown in 1935 by Kaplan[2] who reported a case of carcinoma of the breast in one of homologous twin sisters. Peyron and Kobozieff[3] in 1937 collected from the literature 98 pairs of twins with various tumors. In 23 pairs of homologous twins, only one twin had a tumor. There were no cases of Hodgkin's disease in their collection, nor proved cases of osteogenic sarcoma.

In the present report two pairs of homologous twins were observed in which only one of each pair had Hodgkin's disease without the other twin being affected, and one pair of homologous twins in which only one twin had osteogenic sarcoma without the other being affected.

REPORT OF CASES

CASE 1. M. M., Italian female, aged fifteen, one of homologous twins (Figs. 1 and 2) was admitted to the Brooklyn Cancer Institute on March 11, 1936, complaining of a swelling of the right side of the neck. Three months previous to admission, she had noticed a swelling on the right side of the neck which grew progressively larger. A month later a similar swelling appeared on the left side. She became easily fatigued and had lost 5 pounds within a month.

Her past history revealed that both she and her twin sister had measles, chicken pox and right acute otitis media at about the same time. Both had a tonsillectomy and right mastoidectomy performed. On one occasion the patient developed an abscess of the face and within a few days her twin sister developed an abscess of the thigh. The menses of both twins began at the age of fourteen. Both twins liked baseball and swimming and spent a great deal of time at these sports.

FIG. 1. Case 1. Showing Hodgkin's disease of the neck.

Her mother had received roentgen therapy for Hodgkin's disease at Grace Hospital, Detroit, in 1925, after the diagnosis had been made from an excised gland in the neck. She died in 1928. The patient's father and twin sister are in good health.

* From the Brooklyn Cancer Institute, Division of Cancer, Department of Hospitals, New York City.

Physical examination revealed a well nourished white female, not acutely ill, not in pain, temperature 99.5° F., pulse 100, respiration 24, blood pressure 122/58. A soft boggy tumor was found encircling the anterior surface of the neck, filling both supraclavicular and infraclavicular spaces. The tumor consisted of a conglomeration of freely movable glands, loosely attached to each other in grape-like formation, not attached to the skin or underlying structures. Similar tumors were found palpable in both axillae and inguinal regions, varying in size from a small cherry to a walnut. The chest did not reveal any clinical evidence of a pathological condition. The abdomen was soft. The spleen and liver were not palpable. There were no other masses palpable.

Fig. 2. Case . Showing patient and homologous twin sister.

The urine was normal. Blood chemistry was within normal limits. Blood Wassermann was negative. The blood count showed 3,600,000 red cells with a hemoglobin of 65 per cent. There were 15,950 white cells, with 78 per cent polymorphonuclear leukocytes, 18 per cent lymphocytes, 3 per cent monocytes, and 1 per cent eosinophiles. Sedimentation time was fifteen minutes; coagulation time four minutes. Basal metabolism was minus 35.

Roentgen examination revealed a mass extending from the upper mediastinum into the left apex, and a marked scoliosis of the lumbar vertebrae with a convexity directed to the left side, and a corresponding scoliosis of the dorsal spine with a convexity toward the right. A similar and symmetrical scoliosis of the spine was found on roentgen examination of the twin sister, but the mediastinum was free from any pathological condition.

Roentgen examination of the long bones revealed an area of increased luminosity in the lower third of the right femur, measuring 1 × 1 cm. The skull was normal.

A gland from the neck was excised for histological examination and reported by Dr. Polayes, the pathologist, as follows:

Macroscopic Examination: "Specimen consists of a matted, lobulated mass of lymph nodes measuring 5 cm. in longest diameter. The capsule is thickened. Consistency is firm and the cut surface presents a pale yellow appearance containing numerous small faint tubercles. Smear for acid-fast bacilli is negative."

Microscopic Examination: "The tissue consists of a highly cellular granulomatous process in which there is extensive replacement of the lymph node architecture with fibrosis of the capsule and trabeculae. The cellular infiltrate shows a remarkable degree of pleomorphism, polymorphonuclear leukocytes, including many eosinophiles, plasma cells, lymphocytes, fibroblasts, round cells, and giant cells, together with hyperplasia of the reticulo-endothelial cells. Of the giant cell forms, both mononuclear and multinuclear types are present in which the chromatin masses take a deep stain, and not infrequently the cells are surrounded by a halo with the nuclear characteristic of the Dorothy Reed cell. Some areas reveal considerable fibroblastic activity with productive connective tissue. The marked pleomorphic character of the cell with the presence of many eosinophiles and the presence of Dorothy Reed cells are all highly characteristic of *Hodgkin's lymphogranuloma.*"

The patient remained under our care for seventeen months, receiving several courses of roentgen therapy and transfusions. Another gland was removed from the axilla, which further confirmed the diagnosis of Hodgkin's disease.

During this time she developed herpes zoster over the left chest and a marked itching of the skin associated with jaundice and a marked secondary anemia, with a red count of 1,420,000, hemoglobin 39 per cent, and a white count of 2,350 with 80 per cent polymorphonuclear leukocytes and 20 per cent lymphocytes.

The abdomen became very much distended with fluid. After paracentesis numerous tumors were felt through the abdominal wall. Similar tumors were felt in the pelvis on rectal examination.

The patient died on July 28, 1937.

Case II. A white male, aged fourteen, one of homologous twins, was admitted to the Brook-

lyn Cancer Institute on May 29, 1936, complaining of a swelling on the right side of the neck (Fig. 3). He first noticed the swelling in August, 1934.

Family History. The patient's twin brother was in good physical condition. There was no evidence of any glandular enlargement. Both parents were alive and well, as was a five year old sister. There was no history of Hodgkin's disease in the family, although the mother stated that as a child she had "swollen glands" which, however, disappeared as she grew older.

The mother had a severe case of toxemia during her twin pregnancy with a marked hypertension and nephritis. Labor had to be induced several weeks before term. The patient was the first to be delivered manually, but both infants were in good physical condition. Both twins had mumps, measles, whooping cough and chicken pox at the same time. Both had hypertrophied tonsils and adenoids, which were removed surgically. They had both been subject to hay fever for four or five years. Both were covered with freckles (Fig 4).

Fig. 3. Case 11. Showing Hodgkin's disease of the neck.

Fig. 4. Case 11. Showing patient and homologous twin brother.

Examination revealed a well developed patient, weighing 129 pounds; temperature 100° F., pulse 80, respiration 20. A large firm tumor was found on the right side of the neck, overlying the upper two-thirds of the sternocleidomastoid muscle, encroaching partially on the mandible and side of the face. The tumor was firmly attached to the skin and firmly adherent to the underlying structures. The right supraclavicular space was filled with freely movable glands varying in size from a cherry to a walnut. The rest of the physical examination did not reveal any pathological condition.

The blood count showed 4,440,000 red cells, hemoglobin 60 per cent, and 9,900 white cells, with 89 per cent polymorphonuclear leukocytes, 9 per cent lymphocytes and 6 per cent eosinophiles. Blood Wassermann was negative. Blood chemistry showed urea 23 mg. per 100 cc., creatinine 1.18 mg. per 100 cc., sugar 100 mg. per 100 cc. Urine showed 1 plus albumin.

A gland from the neck was excised and reported by Dr. Polayes as follows:

Macroscopic Examination: "Specimen consists of two lymph nodes, the larger of which is 1 cm. in diameter, encapsulated, and on section made up of smooth yellow-pink homogeneous tissue."

Microscopic Examination: "Section of lymph node shows moderate productive fibrosis with hyalinization of both capsule and trabeculae. The lymph node architecture is considerably altered and replaced by a granulomatous proc-

FIG. 5. Case III. Showing patient whose leg was amputated for osteogenic sarcoma, and his homologous twin brother. A metastatic nodule can be seen over sternum. Both twins had female distribution of pubic hair.

ess. The essential feature throughout is the marked pleomorphism of type cell together with areas of connective tissue proliferation with hyalinization. There are numerous fibroblasts, epithelioid cells, round cells, polymorphonuclear leukocytes and eosinophiles present throughout. An occasional large cell, usually mononuclear and containing abundant chromatin is observed, which is suggestive of the Dorothy Reed cell. Diagnosis: *Hodgkin's lymphogranuloma.*"

Roentgen examination of the lungs on April 26, 1937, was reported by Dr. Westing, the roentgenologist, as follows: "Considerable widening of the supracardiac shadow into the right lung field. The pathological shadow was well defined convex outline and the whole appearance is very suggestive of Hodgkin's disease of the mediastinum."

Roentgen examination of ribs, skull, long bones, pelvis, spine and mandible revealed no evidence of a pathological condition.

During his stay in the hospital the patient received several courses of roentgen therapy and repeated transfusions. Later the patient returned to his home, where he developed a marked abdominal ascites with evidence of abdominal Hodgkin's disease. He died on November 23, 1937.

At this writing the two living twins have been observed for four years two months and five and a half years respectively. They are still enjoying good health, free from any evidence of Hodgkin's disease.

CASE III. A white male, aged sixteen, one of homologous twins (Fig. 5), was admitted to the Brooklyn Cancer Institute on February 26, 1933, complaining of cough, hemoptysis and hoarseness of four months' duration. About a year previous to admission he had developed pain in the upper third of the left leg, close to the knee, and noticed for the first time a swelling in that region, the size of an orange. He was admitted to a hospital on March 1, 1932, where two weeks later his left leg was amputated above the knee for osteogenic sarcoma of the fibula (Fig. 5). He was discharged in June, 1932, and was referred to our institution for further treatment. He and his twin brother had had measles and diphtheria at the same time. His parents, a sister and a brother are living and well.

Examination revealed a sixteen year old male, somewhat undernourished, supporting himself with the aid of crutches, comfortable, not in pain. Temperature 103° F., pulse 128, respiration 22, and blood pressure 130/70. Several shotty glands were palpable in the submental region. The veins of the right side of the neck and the right side of the chest were very prominent. A firm, hard nodule measuring 2 cm. in diameter was found a little to the left of the xiphoid process (Fig. 5). The right upper chest showed evidence of diminished expansion, increased vocal fremitus, and increased dullness with absent breath sounds. Numerous scattered moist râles were present throughout the rest of the lungs. The heart showed evidence of a systolic murmur at the aortic and mitral area. The area of cardiac dullness was increased; the apex extended to the sixth interspace. The abdomen was soft, the liver edges extended two fingerbreadths below the costal margin. The spleen

Fig. 6. Case III. Roentgenogram showing metastatic osteogenic sarcoma of the left lung.

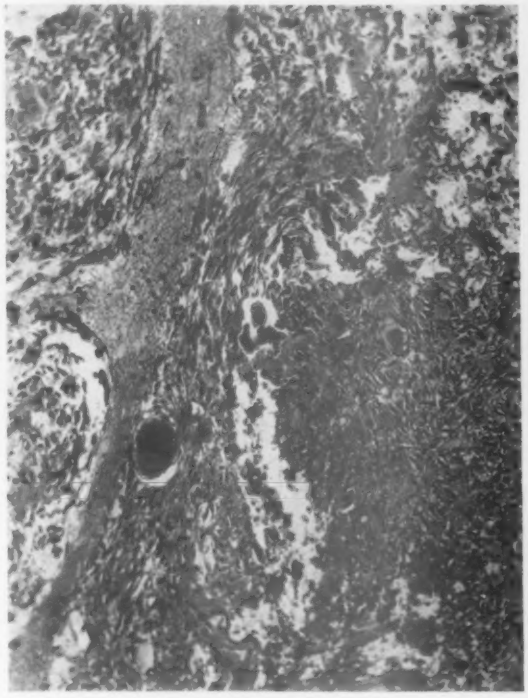

Fig. 8. Case III. Photomicrograph (×200) of osteogenic sarcoma showing sheets of spindle cells, scattered giant cells, and spicules of new bone.

Fig. 7. Case III. Roentgenogram showing p imary lesion of osteogenic sarcoma of the upper end of the fibula.

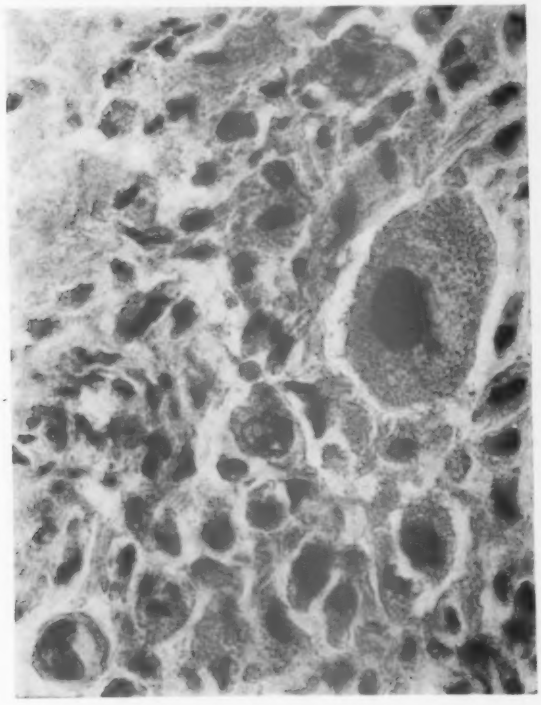

Fig. 9. Case III. Photomicrograph (×500) of osteogenic sarcoma, showing anaplastic characteristics of tumor cells.

was not palpable; no other masses were palpable. The inguinal glands were not enlarged. There was a female distribution of pubic hair (the same distribution was found in the twin brother).

The left lower extremity had been previously amputated at the junction of the upper and middle third of the thigh, and the stump was completely healed without any evidence of recurrence. The rest of the extremities did not show any pathological condition. No pelvic masses were palpable on rectal examination.

Urinalysis was negative. The blood chemistry was within normal limits; the blood Wassermann was negative. The blood count showed 4,000,000 red cells with 65 per cent hemoglobin. The white count was 10,350, with 72 per cent polymorphonuclear leukocytes and 28 per cent lymphocytes. Roentgen examination of the chest revealed a large tumor involving the upper two-thirds of the right lung field (Fig. 6). Roentgen examination of the long bones and pelvis showed no evidence of a malignant lesion.

Following roentgen therapy to the lungs and general supportive treatment, the patient was taken to another hospital, where he died on April 19, 1933. An autopsy revealed metastatic osteogenic sarcoma of the lung, pericardium, pleura, heart (ventricles and auricles), diaphragm, anterior chest wall, left rectis muscle, kidneys, renal veins, and right iliac veins.

Since the patient's admission to the Brooklyn Cancer Institute on February 26, 1933, up to the present writing (seven years) his homologous brother (Fig. 5) has been closely observed. He has been examined every three months, including roentgen studies of both extremities. Up to the present writing, eight years since his brother's illness, he is enjoying good health and shows no clinical or roentgenological evidence of malignant disease.

SUMMARY AND CONCLUSIONS

1. The study of homologous twins would seemingly contribute a great deal toward solving the genetic theory of cancer, if sufficient clinical material were available for study.

2. Tumors occurring in homologous twins do not always seem to be similar, symmetrical and simultaneous, although they did so occur in the majority of cases reported in the literature.

3. Two cases of Hodgkin's disease and one case of osteogenic sarcoma in homologous twins are described, where only one of each pair of twins acquired the disease. The homologous living twins are now free from any symptoms and physical findings four years two months, five and a half and eight years (Cases I, II and III respectively) since the disease was first recognized in the deceased twin.

4. There are no similar cases reported in the literature.

5. We shall continue to observe these twins in years to come and a further report will be made should they show any evidence of Hodgkin's disease or osteogenic sarcoma.

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SOME CASES OF SWALLOWING OF MISCELLANEOUS OBJECTS*†

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WE HAVE recently had occasion to observe some cases of swallowing of miscellaneous objects, without doubt interesting particularly from the point of view of roentgen iconography and on account of the great number and variety of the foreign bodies swallowed by the patients.

It is certainly not unusual to find various objects in the digestive tracts of certain individuals, particularly children, mental defectives and prisoners; medals, coins, pins or pieces of iron may be said to be found frequently, but the swallowing of large numbers of bodies is rare and in recent years we have had 3 of these cases: one a prisoner who swallowed the most varied sorts of objects in order to have himself admitted for a time to the hospital, a peasant who had swallowed nails for the purpose of suicide and a man, certainly not brilliant mentally, who performed such acts for purposes of exhibitionism, without thinking of the danger to which he was subjecting himself, to such an extent that finally his activity resulted in death.

CASE 1. This patient was a male, S. S., aged twenty-four, farmer, admitted January 17, 1936, in the first surgical section (Chief, Dr. D'Este). He said that two months before he had swallowed a large number of nails for the purpose of suicide. At first he had a sense of pain in the epigastrium, a feeling of weight and moderate loss of appetite, but these disturbances gradually decreased until he could eat normally. But for the past three weeks he had had pain in the stomach again, accompanied by vomiting. He was constipated.

The general condition of the patient on admission was good: color normal, tongue moist, slightly coated.

Objective examination of the abdomen showed a certain amount of pain on palpation in the epigastric region. Nothing else of importance in the various organs. But the finding of areflexia of the pharynx was important.

A roentgen examination on January 20 revealed a large mass of nails in the abdomen, all in the stomach, so that the form of the stomach was perfectly reproduced; they were almost all arranged in the direction of the curvatures and folds of the stomach mucosa. No nail had passed into the intestine (Fig. 1).

FIG. 1. Case 1. The stomach is full of metallic nails which show its form. No nail has passed into the intestine after two months.

The necessary surgical operation performed on January 21 (Chief, D'Este) led to the extraction of 72 nails, some as much as 8 cm. long, and three pieces of iron, weighing altogether 293 gm. The wall of the stomach was hypertrophied, the serosa opaque in some places, especially in

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† Translated from the Italian by Audrey G. Morgan, M.D., Los Angeles, California.

Fig. 2. Case II. Some fragments of glass are distinctly visible in the descending colon which is full of gas.

the antral region, the mucosa very red and hypertrophied. When freed of the foreign bodies the stomach contracted rapidly.

After a normal postoperative course the patient was discharged cured on February 2, of course after a control roentgen examination which did not show any foreign bodies remaining in the digestive tract.

Case II. M. G., a baker, aged twenty-one, of Wiener Neustadt, a prisoner was sent from the jail to the hospital and admitted to the first surgical section (Chief, D'Este), in January, 1936, after having swallowed bits of glass and a metal buckle for the purpose of suicide (?).

On admission he complained of abdominal pain and attacks of vomiting. He was in good general condition, apyretic, tongue moist, moderately clean. Objectively there was pain in the epigastrium and in the left lower quadrant of the abdomen, especially marked on pressure.

During his stay in the hospital a roentgen examination was made which showed the presence of foreign bodies, many of them in the large intestine. Successive roentgen examinations showed the progression and gradual elimination of the fragments of glass (Fig. 2).

The patient was then discharged without operation, which the surgeons did not think was indicated. During his stay in the hospital he had attacks of convulsions which were thought to be simulated.

After some months, in June, this same individual was sent to the hospital again, this time also from prison, for having swallowed other objects. Objective examination this time showed only a certain amount of pain along the course of the colon.

Roentgen examination showed many irregularly rounded opaque bodies along almost the whole course of the large intestine, and fragments of objects of metallic opacity (Fig. 3).

After two weeks he complained of very violent pain and febrile temperature, while he continuously evacuated stones. Gradually there was improvement, though he continued to evacuate bits of metal (Fig. 4).

After about forty days in the hospital his general condition was good and there were no signs of visceral lesions; in view of his psychic condition and behavior and in the absence of abdominal symptoms he was sent back to prison.

Fig. 3. Case II. The transverse colon is full of rounded opaque bodies, some of which are already in the descending colon.

In September of the same year he was again admitted with violent pain in the stomach, hematemesis and melena and febrile temperature. Objectively, there was abdominal rigidity, particularly in the epigastric region. He had swallowed foreign bodies again.

Roentgen examination showed the presence of metallic bodies in the digestive tract. A large one, apparently a coil of metal wire, was in the cecum. In a later control examination the large metallic body had progressed and was found in about the middle of the transverse colon, while other metallic bodies were visible (Fig. 5). After some days these objects also were eliminated naturally.

The last roentgenogram of this patient was taken in 1938, after he had been admitted again in 1937 for again having swallowed foreign bodies. In this roentgenogram also the metallic foreign bodies were very evident in the large intestine, while the stomach was full of barium paste (Fig. 6). This time also the result was natural evacuation.

Up to the present time (February, 1939) we have not seen the patient again.

Fig. 4. Case II. After the elimination of many stones, bits of metal are visible in the transverse colon. Some stones still remain in the cecum, ascending, transverse and descending colon.

Fig. 5. Case II. Large metallic bodies in the ascending and transverse colon.

Case III. F. M., a laborer, aged forty-seven, married; he came on September 14, 1936, to the clinic of the Regina Elena Hospital, saying that while drunk he had swallowed on a wager three stylographic pens, a bunch of five keys and various other objects.

Fig. 6. Case II. Other metallic bodies are present in the large intestine. Their presence in the stomach is excluded by the administration of opaque paste.

Fig. 7. Case III. The stomach is full of the most varied foreign bodies, which outline its form. No object has passed into the small intestine.

Sent to our Institute for roentgen examination many foreign bodies of the most varied kinds were found in his stomach (Fig. 7); the patient himself presented a list of almost 80 of these bodies. During the examination he vomited a pencil. As a result of this finding he was sent to the surgical section (Prof. Oliani) still under the influence of alcohol.

Physical examination showed an individual evidently under the influence of alcohol. A certain amount of resistance was palpated in the epigastrium which was slightly sensitive on palpation. The fauces were intensely red and there were some excoriations on the hard and soft palates.

Two days later surgical operation was performed (Prof. Oliani). Median supra-umbilical laparotomy, gastrotomy. Seventy-five objects were extracted, including stones, keys and stylographic pens (Fig. 8).

Two weeks after the operation the patient was discharged cured with certain instructions which, however, he did not follow, as he was readmitted to the hospital on January 16, 1937, complaining of abdominal pain and stating that on the last feast day he had swallowed various objects for the purpose of exhibitionism, such as coins, spoons, a watch, etc.

Objectively he was in good general condition, afebrile with pulse regular and rhythmical. There was abdominal rigidity, most marked in the right quadrants, where palpation was very painful. There was also marked meteorism.

Roentgen examination showed the presence of foreign bodies (Fig. 9) which necessitated surgical operation (Prof. Oliani). When the peritoneum was opened pus was discharged; the serosa was red and covered with fibrinous tracts. In one loop of the small intestine a coffee spoon was palpated; the foreign body, 10 cm. long, was extracted through a transverse incision. Two other spoons as long as this one were found free in the peritoneal cavity; they had passed out of the intestine through a lacerated perforation about 5 cm. long at the duodenojejunal angle. The lesion was sutured and antiperitonitis serum injected into the peritoneal cavity.

Fig. 8. Case III. The objects extracted from the stomach are lined up on the operating table. There are many pens, keys, coins and various objects.

Fig. 9. Case III. Three spoons are visible (two of them superimposed) in the abdomen. Marked meteorism, presence of large collections of gas and liquid.

The patient's general condition grew progressively worse and he died on the thirteenth day with a temperature of 36° C. and a pulse of 120.

DISCUSSION

After having read the histories of these 3 patients and seen the roentgenograms, little remains to be said and little to be considered. It can be seen that even large objects can pass through the esophagus without excessive difficulty, enter the stomach and, after a more or less prolonged stay, pass the whole intestinal tract to the rectum and be eliminated readily. The chief obstacle to their progress is the pylorus, or when the objects are long and rigid, the narrow curves of the duodenum, particularly the lower curvature between the second and third portions of the duodenum, and the duodenojejunal angle, an obstacle which proved fatal in our third case.

The exact localization of the object by roentgen rays may sometimes prove difficult, but the progress of the object through the intestine gives definite information, particularly with the aid of opaque paste. The symptoms complained of by the patient may vary greatly, depending on the number and nature of the objects swallowed; there is always danger of decubital ulcer or perforation if the object remains for a long time at one point in the digestive tract, resulting in perforation peritonitis or circumscribed abscess which may open externally, but this is rare, even with cutting objects, and hemorrhages are therefore not frequent.

I will not discuss the treatment of such conditions at length; usually the foreign bodies are eliminated spontaneously, but surgical operation becomes necessary if they remain fixed too long at one point; the urgency of operation in cases of perforation is obvious.

We have found described in the literature 2 cases in which two pins were found, probably introduced through the mouth, which had followed the bile tracts backward and reached the gallbladder. Roentgenologically by the administration of opaque paste and then of tetraiodide it was possible to make an accurate diagnosis of the localization and in one of the cases this was confirmed by operation.

We mention this case as a curiosity; and the cases presented in this brief note may be considered interesting from that point of view also.

END-RESULTS IN IRRADIATED HODGKIN'S DISEASE*

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MORE than a century has passed since Thomas Hodgkin[9] reported several unusual cases of lymph node enlargements representing the peculiar and rare disease which Wilks[20] described more accurately in 1856 and later referred to as Hodgkin's disease. Little progress has since been made in solving the problem it presents.

Hodgkin's disease will be used here to describe that particular clinical syndrome, febrile or afebrile in its acute or chronic but usually fatal course, which has early or late a fairly definite peripheral blood picture and objectively has its beginning in one or more lymph nodes or some part of the auxiliary lymphoid system. It may remain localized for a long time or become generalized, presenting a rather characteristic histopathologic pattern consisting of proliferations of the reticular and endothelial cells, formation of lymphoid cells, characteristic giant cells and a gradual increase of connective tissue resulting in fibrosis. Since it appears to be a disease of the hemopoietic organs, viz. bone marrow, lymph nodes, spleen and liver and may make its appearance in almost any tissue, there is little wonder that its manifestations are protean.

A voluminous literature has accumulated about its nature and etiology. In an earlier paper[14] some of the controversial aspects were reviewed. Opinion today is no less divided. There are those who incline to the concept that the disease is of infectious origin, doubtless influenced by the early work of Sternberg,[19] who in 1898 published a monograph treating Hodgkin's disease as a form of tuberculosis which view he later abandoned. Others feel supported in their contention that the disease is an inflammatory process by the work of Gordon[8] whose findings permit the assumption of a specific infectious agent. Renewed interest in its possible infectious origin has been aroused by Parsons and Poston[16] who recently reported 4 cases of *Brucella* infection in which the histopathology was identical with that of Hodgkin's disease. On the other hand, numerous cases known from the literature lend support to the belief that all clinical and pathological varieties of Hodgkin's disease may be regarded as a neoplastic development in the reticular components of the hemopoietic system.

Hodgkin's disease may be considered acute (lasting a few weeks to a few months); subacute (from a few months to two years); chronic or average type (from two to six years) and protracted (over six years). This is the clinical classification suggested by Gilbert and his associates[7] and is useful from a statistical viewpoint. There is, of course, no pathological basis for a clinical division into acute and chronic forms, since both exhibit the same morphological changes but with a different chronological spacing.

While we most often think of Hodgkin's disease as one involving the peripheral lymph nodes or the spleen and liver it should not be forgotten that the primary focus, at least at the time of discovery, may be elsewhere. Reeves[17] found 7 cases which began in the tissues of the orbit. Blount[2] reported a case in which involvement of the spine and left shoulder preceded generalized lymphadenopathy. In our cases primary lesions were found in the nasopharynx, the thyroid, the mediastinum, the stomach, the abdominal lymph nodes, the mesentery, the skin and epidural space with prodromal symptoms as diverse as otalgia, deafness, enlargement of the thyroid, dyspnea, indigestion, abdominal pain, jaundice, itching, sensory changes and cord block.

Some anemia of the secondary type is a regular accompaniment of this disease. Cer-

* Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

tain authors describe it as a progressive anemia. It would be more accurate to state that the anemia becomes progressive as the terminal stage of the disease approaches. For long periods in the average and protracted types the erythrocytes and hemoglobin remain close to normal. The total leukocyte count is usually within normal limits or slightly above. As the disease progresses there is an increase in total leukocytes to about 20,000 with a low lymphocyte and high polymorphonuclear percentage. This blood picture may appear at the onset in the acute and subacute cases.

Several procedures have been offered as an aid in diagnosis. In the presence of a mediastinal tumor without evident adenopathy when biopsy is obviously impracticable, so-called therapeutic irradiation is often counselled as a differential diagnostic test on the principle that lymphoid tissue because of its high sensitivity to roentgen radiation will recede promptly. A dosage of 600 r is often prescribed. This may be effective when given at one sitting but inadequate when fractionated and so prove

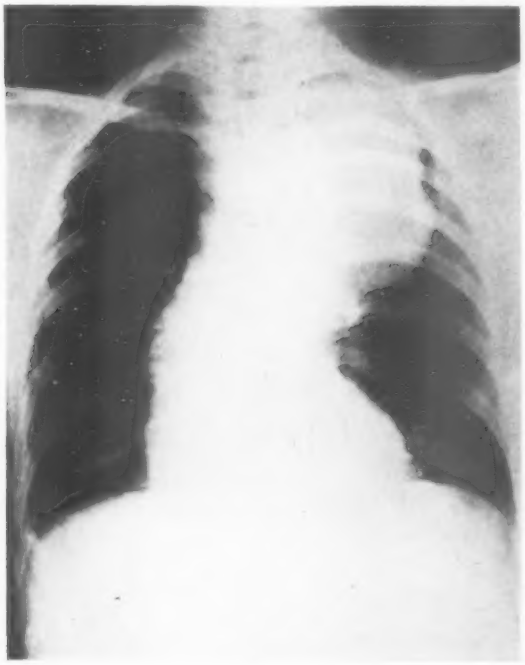

Fig. 1

Fig. 2

fallacious as a diagnostic test if rigidly adhered to. Furthermore, this maneuver should be used with caution. Aneurysm and tuberculous mediastinitis may simulate Hodgkin's disease of the mediastinum. I have made the roentgen diagnosis, corroborated by others, of mediastinal lymphoma in a patient who died from aneurysmal rupture as he was preparing to leave his home to receive irradiation (Fig. 1). One other patient had Hodgkin's disease by biopsy but died of miliary tuberculosis following irradiation of mediastinal and lung root masses (Figs. 2 and 3).

Gordon[8] in 1932 demonstrated that intracerebral inoculation of rabbits with a sterile emulsion of Hodgkin's nodes was followed by the appearance of an encephalic syndrome which observation is the basis of the so-called Gordon test. It has been shown by later investigators, however, that it is the eosinophiles in the Hodgkin's nodes which are responsible for the encephalic reaction in the rabbit and that it can be produced with other tissues containing those cells. While it may be considered a confirmatory test, being positive in 69 to 85 per cent of cases of Hodgkin's disease, it is

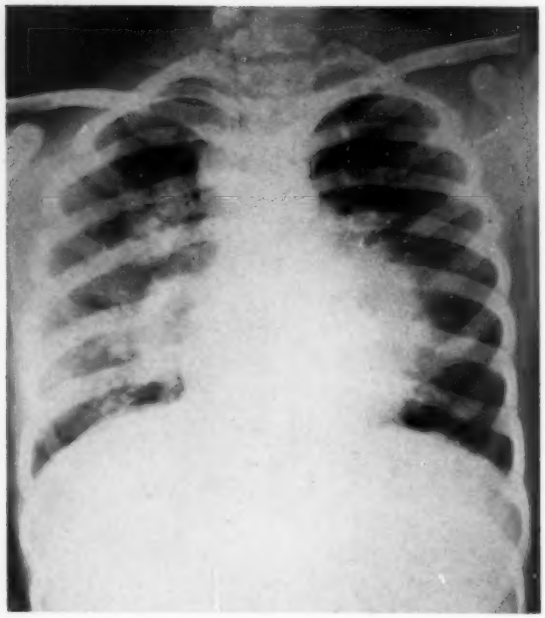

Fig. 3

not specific. Study of the bone marrow also has been advocated by others but the results do not appear to have been conclusive or even useful.

The diagnosis of Hodgkin's disease based on anything else but histological evidence is to be deprecated. Surgical excision is the method of choice. Aspiration biopsy here is rarely of value. Biopsy should be done promptly for early diagnosis may alter radically the treatment which might otherwise be instituted. Regression in the size and number of objective nodes is common without any formal therapy which accounts in many instances for the delay in arriving at a correct diagnosis. Thirteen, or 21.6 per cent, of the cases in our series had the disease on the average almost four years before biopsy was done. Biopsy is equally valuable in allocating to proper place and appropriate treatment those cases which simulate Hodgkin's disease clinically.

The therapeusis of this disease for the most part has consisted of the arsenicals, blood transfusion, surgery or irradiation, often a combination of all with the occasional isolated use of heliotherapy.

Surgical excision of localized nodes with and without irradiation seems definitely to have influenced the duration of life of some cases of genuine Hodgkin's disease. Wright[21] records the case of a male aged fifty-nine who had surgical removal of Hodgkin's nodes twice within a year followed by high voltage irradiation and no subsequent treatment, and eleven years later was alive without recurrence. Baker and Mann[1] cite 3 cases alive thirteen, eleven and nine years following surgical dissection of localized Hodgkin's nodes. One case operated on in 1929 in which a mass of nodes was removed from the posterior triangle of the neck showed at the time roentgenological evidence of mediastinal involvement. He was given deep roentgen therapy and although various nodes and even the spleen have been enlarged at times he was in good general health nine years after.

One case in our series, a male, is alive and clinically free from disease twenty years after surgical excision of cervical nodes in 1920. He lived for nineteen years without recurrence. Supraclavicular nodes were then excised (1939) in the presence of a

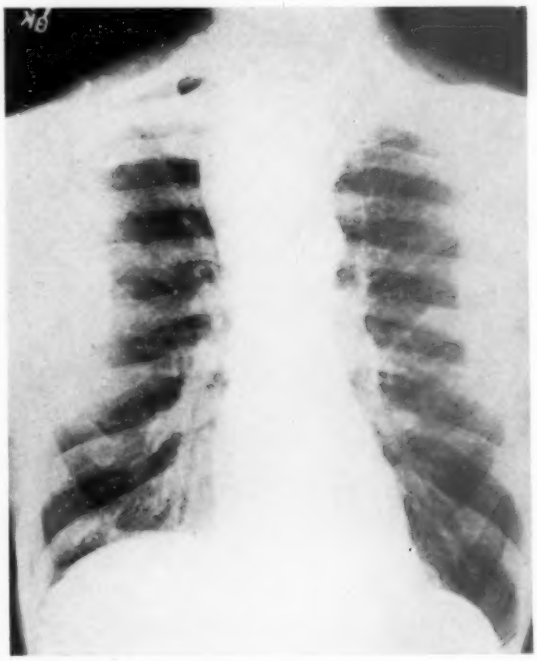

Fig. 4

known mediastinal (Fig. 4) tumor by the surgeon who performed the original excision. After high voltage roentgen therapy the mediastinal tumor was no longer demonstrable. The pathological report was as follows: "Hodgkin's granuloma showing more fibrosis than previous specimen reported in 1920."

The majority of cases seen are not favorable for surgery. Irradiation (most commonly by roentgen rays) continues as the bulwark of treatment. The method of irradiation is not at all standardized. Some treat the objective localized lesions only with small doses that are fractionated, or with relatively large doses at one sitting. Then there are those who advocate total body irradiation through large portals, systematically irradiating the obvious lesions and the possible sites of spread. Still others employ teleroentgen therapy where one surface of the entire body is subjected to irradiation at each seance.

Any or all of these methods may be found useful and their employment should be governed by the stage of the disease, the age and general condition of the patient. In a child presenting enlarged nodes confined to the neck without biopsy in whom the diagnosis of tuberculous adenitis is in abeyance one would be rash to give intensive roentgen irradiation. Yet if the biopsy is positive for Hodgkin's disease one would be justified in using large doses to a localized group of nodes with the hope of fibrosing them and delimiting the process.

Most of the patients who have lived longest have had the disease localized over a long period of time before the general lymphatic system became involved. Even though the theory is accepted that Hodgkin's disease is systemic there is no evidence that irradiating the trunk or whole body has meant prolongation of life for the entire group. The absolute survival rate should be the norm here as elsewhere.

In the presence of fever and prostration it does not seem rational to use large doses or a formulated spatial distribution that will include the whole body sectionally or in toto. Some write enthusiastically about teleroentgen therapy but mostly in academic fashion. Cases are reported in a desultory manner, few having been followed long enough to warrant any conclusion but that it is an ancillary method with definite limitations. Marchal, Mallet and Bellin[13] report 24 cases of Hodgkin's disease verified by biopsy so treated that in half "brilliant and durable" results were obtained. Seven of these 12 cases had no other treatment besides teleroentgen therapy. One patient has lived for seven years and one for eight. Details regarding the others are lacking. They warn that teleroentgen therapy may produce marked inhibition of the bone marrow and that the prevention of untoward reactions requires repeated and careful blood examinations in the course of the treatment, best performed before every irradiation. At the Boston City Hospital teleroentgen therapy has been found more useful in the chronic leukemias. In Hodgkin's disease it has always been necessary to supplement it by focal treatment of the spleen and other large lymphoid masses.

Usually the erythrocytes are not affected adversely by irradiation. It is common to see a rise in hemoglobin following irradiation. A falling hemoglobin during irradiation usually means the terminal stage of the disease with one exception and that is when teleroentgen therapy is employed injudiciously. Lymphopenia often promptly follows even small fractionated doses of irradiation and need not be a contraindication. The use of intensive doses and large portals, however, may decrease the leukocytes to a dangerous level.

Pulmonary fibrosis must be considered among the possible and undesirable sequelae of irradiation. It is difficult to evaluate. A generalized fine pulmonary fibrosis is a physiologic change common in adults. It may also represent an invasion of the peribronchial lymphatics by Hodgkin's disease. When it is known that fibrosis is the end-result in the evolution of Hodgkin's dis-

ease it is not easy to dogmatize regarding changes seen in the lungs.

Mallory[12] has described a solid fibrosis of an upper lobe in Hodgkin's disease where it was difficult to rule out irradiation as the cause (Fig. 5). So rare is a detailed description of postmortem findings of Hodgkin's disease in the lung after irradiation that I am constrained to quote it at some length.

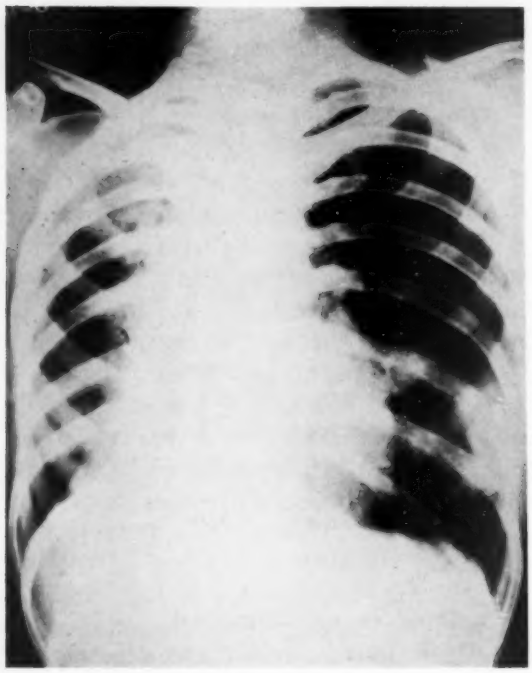

Fig. 5

One of the most striking features was the right upper lobe which was completely lacking in air and was of a uniform leathery consistence. I think the degree of dense fibrous consolidation in the upper lobe is not indicated adequately in any of the films. It had shrunken enough so that the apparent apex of the lung consisted actually of the apex of the lower lobe. Aerated lower-lobe tissue lay behind the upper lobe at all points, so that in the X-rays we get the appearance of at least some air in the apex of the lung, whereas there actually was none in the upper lobe.

Microscopic examination showed solid fibrosis of this entire lobe with extremely dense collagen and just an occasional remnant of an alveolus line with cuboidal epithelium. Throughout this fibrous mass, however, one could still recognize persistence of alveolar walls. The elastic tissue had not been destroyed and there was an alveolar pattern that could be brought out with the elastic-tissue stain but which was quite invisible with the hematoxylin and eosin stain. Where we found recognizable Hodgkin's involvement of the lung in this case, there was always complete destruction of the lung architecture. In the upper lobe there was fibrous obliteration of all the air sacs but the architecture was not obliterated. Either this was once Hodgkin's disease and all the Hodgkin's cells were killed or sterilized by X-ray so we could no longer recognize them, or we must assume that this fibrosis of the entire lobe was due to something else. I can only guess what that something else might be. The largest mass of nodes was in the lower right neck and was very resistant to therapy. She had a great many treatments to the mediastinum and chest. Therefore there was possibility of a good deal of cross-firing, and much of the radiation that she had may have accumulated in this one area. The experimental production of X-ray pneumonitis has never been carried to such an extent as to produce anything like this picture. I cannot say that it has ever been proved that radiation pneumonitis can consolidate a lung to this extent, but I think it is more likely that the changes were produced in that way than that they were a part of the Hodgkin's involvement.

Until more is learned about post-irradiation changes it would appear to be the part of wisdom to exercise caution against piling up large doses of radiation in lung tissue, either directly or by cross-firing or both.

At the Boston City Hospital since 1933 we have used high voltage (200 kv.) roentgen radiation exclusively in our treatment of Hodgkin's disease because it allows of a flexibility not practicable in voltages of lower ranges. Whether it is desired to use small or large doses, of low or high intensity, small or large portals, at short or long distances, such a potential makes it feasible with proper filtration to repeat courses of irradiation without damage to the skin, not possible otherwise.

One should not confuse the use of high voltage radiation as employed in the treatment of Hodgkin's disease with its employment in carcinoma where the total dose

may reach 5,000 r given in fractions of 200 r or more. Seldom should one in treating Hodgkin's disease exceed a total fractionated dosage of 2,000 r in one course. Such a dosage should be approached with prudence. Occasionally splenomegaly and mediastinal masses will not respond to less. Apropos of small doses as against large doses, Spritzer[18] reports a series of 27 patients treated from 1926 to 1932 with small doses of 200 to 600 r, repeated every few months, whose average duration of life from the first treatment was 2.5 years and a series of 29 patients treated from 1932 to 1936 with large fractionated doses of 800 to 5,000 r who lived only 1.5 years after treatment.

It is our custom routinely to fractionate the dose using 150 to 200 r for localized nodes, mediastinum or spleen, with relatively small portals. No attempt is made to treat possible hidden foci by sectional irradiation of the body. When it is apparent that the disease is generalized and larger portals are indicated an initial dose of 50 r is given every other day, graduated with care until 150 r is reached, similar to the regional thoracic and trunk baths described by Levitt.[11] When we have employed teleroentgen therapy (focus-skin distance 12 feet), exposing an entire surface of the body to irradiation at one time, 10 r delivered at the center of the field has not been exceeded.

Treatment of necessity must be highly individualized. Choice of irradiation factors is governed by the general condition of the patient and extent of the disease. No case is accepted for radiation treatment unless properly worked up. Each patient is seen regularly and followed by the medical service having referred it and the amount of radiation given is controlled by their clinical observations.

From 1919 to 1939, 116 cases were referred to the Boston City Hospital for radiation therapy with the diagnosis of Hodgkin's disease. Twenty-three were clinically Hodgkin's but had no biopsy. Four were positive on biopsy but were acutely ill and received no irradiation. Eleven on review of the sections were placed elsewhere in the lymphoma group. Eight on biopsy or autopsy proved to be carcinoma, 7 tuberculosis, one a hypernephroma, one myasthenia gravis and one chronic inflammation. On analysis of these 56 cases it is apparent that over 55 per cent were some other condition than Hodgkin's disease. It not only stresses the importance of biopsy but tends to vitiate the value of statistical cures based on a diagnosis of clinical Hodgkin's disease.

Sixty cases of Hodgkin's disease with positive biopsy remain who had the disease on the average of 20.5 months before beginning roentgen therapy and lived on the average nineteen months following this therapy. Five lived on the average nine years and four months after irradiation was begun and one has lived twenty years.

There were 19 females and 41 males in the group, the youngest a female aged fourteen and the oldest a male aged seventy-five. The average age of the females was about forty-one years and the males about twenty-eight years when referred for radiation therapy.

Survival statistics seem futile without an analysis based on the character and extent of the disease when first seen for irradiation. Forty cases, or 66.6 per cent, lived less than two years from the computed onset of the disease and are classified as acute and subacute; 15, or 20.5 per cent, two to six years, as average, and 5, or 8.3 per cent, as protracted. Ten had massive mediastinal involvement, 8 pleural effusion, 4 known tumor in the lung, 6 general adenopathy with abdominal ascites, 6 cord and nerve involvement, 1 generalized skeletal invasion, 2 destruction of multiple vertebrae, 2 localized osseous lesions and 1 Hodgkin's sarcoma of the stomach. It is of some interest to note that there was but one proved case of pulmonary tuberculosis associated with Hodgkin's disease in this series, confirmatory of present opinion that Hodgkin's disease and tuberculosis are

separate and distinct and have no relation to each other.

It is questionable how much is gained by attempting to correlate published statistics many of which contain large numbers of cases unverified histologically. Some use the term survival rate as synonymous with duration of life from the onset of the disease, others mean duration of life from the beginning of irradiation. Some report only selected cases and do not include those lost to follow up.

The ideal would be a comparison of irradiated with a control group of nonirradiated cases. Kasabach and McAlpin[10] have attempted this. Their control group is small, only 7 cases. Their irradiated cases are divided into two groups, one of 20 and one of 38 cases. The first group which they believed had inadequate irradiation lived on the average only two months longer than the control. The second group which in their opinion had more adequate roentgen therapy lived on the average five years and four months after the treatment. An analysis of this group lends itself to the interpretation that there were more of the chronic and protracted types in it.

Craver[3] in 1935 reviewed 125 cases of Hodgkin's disease admitted to the Memorial Hospital between 1917 and July, 1929, and found a five year survival of 12 per cent. The average survival for the group was less than two years. In 1938 he[4] examined 121 cases admitted from January, 1918, to December, 1930, and reported a five year survival rate of 17 per cent. There was no break up of these cases into clinical types or average survival stated.

In 1937 Gilbert[6] presented a comprehensive review to the Fifth International Congress of Radiology on radiotherapy in Hodgkin's disease in which he discussed 77 cases (18 per cent not verified by biopsy) treated by himself and his associates. Out of a group of 52 cases selected, he says, on the basis of deep roentgen therapy systematically given in daily fractional doses, he has 16, or 30.8 per cent, living with a survival period of six years and four months. In 1934 he[7] reported 17 cases from this same selected group surviving on the average six years and five months after the beginning of roentgen therapy. As I have indicated elsewhere[15] 5 of these lived a total of sixty-nine years and four months which in a group of 17 cases is bound to produce a misleading survival rate. When the living cases are taken out of the selected group and plotted against the total number of cases treated by roentgen irradiation the five year plus survival rate is reduced to a little over 12 per cent.

Craver[5] in explanation of the high survival rate published by Gilbert offers the suggestion that he probably sees more early cases of Hodgkin's disease. A more likely explanation is that Gilbert sees a different clinical type. This is corroborated by his statistics for in his selected group which shows the highest survival rate 82.7 per cent are of the chronic and protracted forms.

From an analysis of the 60 cases I am reporting here, and collated statistics from comparable sources, I find no evidence that irradiation has influenced the average duration of life of patients as a group with Hodgkin's disease. Some apparently destined to have the disease for a long time have lived for periods of ten to twenty-five years following surgery, irradiation or a combination of both. Irradiation is definitely life saving for some and helps others live in comparative comfort for a few years. That it transforms a malignant morbid process into one that is benign, thus doubling and trebling the life expectancy of those with the disease as has been suggested does not appear tenable.

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DISCUSSION

Dr. Edward M. Gall, Boston. It has been highly gratifying to learn Dr. O'Brien's views on this particular disease, a disease which has interested us at the Massachusetts General Hospital for a period of years. It is particularly gratifying to hear him make two comments, one regarding the division of Hodgkin's disease into a more and a less malignant type of the disease, and the other regarding that interdicted type or form of treatment, radical surgery.

I will limit my remarks to our observations on these two points in the series we have studied, which consists of 618 cases, all biopsied or autopsied. There were about 250 cases that would come into the Hodgkin's classification. We found that they fell into two very distinct categories, one consisting of the classical Hodgkin's type of lesion, with the so-called multinucleated giant cells, or the Dorothy Reed or Sternberg cells, in association with a polycellular picture; and the other which we have termed Hodgkin's sarcoma, in which the lesion consists of giant cells alone without the polycellular reaction.

If we considered the clinical course of those individuals in these two categories, we found that the average duration of individuals with classical Hodgkin's was 4.3 years; those with Hodgkin's sarcoma almost invariably died before the first year had elapsed. There was, therefore, a definite morphologic and clinical basis for the division of this disease into two groups.

As to the question of surgical intervention in the larger series of cases which included all forms of malignant lymphoma in addition to Hodgkin's disease (i.e., lymphosarcoma, reticulum cell sarcoma, etc.), we found that a certain number of individuals who, by virtue of the histopathology of the lesions would be expected to die in a relatively short period of time, lived for surprisingly long periods; twelve to sixteen years, in some and in one instance, twenty-four years. In this particular group we found a very interesting clinical procedure had been followed.

In the entire group of 618 cases, 75 were approached surgically without knowledge of the underlying disease, and attempts at eradication of the process were made. Of these, 44 were found to be ineradicable from a surgical point of view.

Among the 30-odd remaining cases treated by radical surgery, 7 died immediately following the operation, presumably from operative trauma. Four were lost, after discharge but some 20-odd cases were followed for extended periods until the time they developed recurrence and died, or to the present time in the absence of recurrence. The interesting point was that the majority of these fell into the very malignant types of lymphoma but despite this fact they lived for periods averaging 7.1 years. Of those

developing recurrence, it was found that the first recurrence did not appear until five and a half years after the operation, and since the period of duration of the entire series as a whole was about three years, they lived practically twice as long as individuals who did not receive surgical treatment. This series is extremely small and is not to be construed to advocate radical surgery in all cases of malignant lymphoma but is of considerable interest in that it offers support to the suggestion expressed by Dr. O'Brien.

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THE EFFECTS OF ROENTGEN-RAY DOSAGE IN BREAST CARCINOMA*

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THE purpose of this paper is to describe a small series of 5 cases of breast carcinoma in which large doses of roentgen rays were given and radical mastectomy done subsequently. These 5 cases were selected from many at our disposal because of the comparatively large radiation dose used. Nevertheless, meticulous histological study of all the tissues removed after irradiation showed persistent carcinoma in all.

The radiation factors used in the treatment of these patients were practically identical, although in 2 of the cases the copper filtration was decreased from 2 to 1 mm. The fields varied somewhat in size according to the reaction of the tumor and the skin. The equipment consisted of Waite and Bartlett, 200 kv. (peak) machine. The factors used were: 200 kv., 25 ma., 50 cm. target-skin distance, and a filtration of 1 mm. of aluminum, and 1 or 2 mm. of copper.

The method of histological study of the tissues removed in radical mastectomy deserves special comment. The breast is cut into many thin slices and meticulously studied for gross evidence of disease. Blocks of tissue are taken for sectioning from every area of induration, intraductal proliferation, cyst formation, or other abnormality. In addition, several blocks are taken from the original site of the carcinoma as well as through the nipple, through the duct system deep to the nipple, and from each of the four quadrants of the breast. It is not unusual to cut as many as fifteen or twenty different blocks of tissue for sectioning. In addition, the lymph nodes are carefully dissected out of the axillary tissues and all of them are sectioned. These tissues are fixed in Zenker's fluid and stained by a variety of methods, including hematoxylin and eosin, phosphotungstic acid, mucicarmine, and Masson's trichrome. The latter stain is of special value in finding small groups of carcinoma cells in heavily irradiated tissue. Even with this thorough method of study, we have not always found the detection of persistent carcinoma to be an easy task, and we can well imagine that small foci of persisting disease could escape less thorough search.

CASE REPORTS

CASE 1 (No. 549037). S.S., a white woman, aged fifty-one, was admitted on April 28, 1938, with a history of having had a small lump in the left breast for several years. She had been aware of a slight increase in its size only during the past year.

Local Examination. The breasts were equal in height. In the lower hemisphere of the left breast was found a 6×5 cm., non-tender tumor attached to and retracting the nipple. The skin over the lower portion of this breast showed edema. The breast was freely movable on the chest wall. In the left axilla there was a hard, 2×1 cm., freely movable lymph node. There was no evidence of distant metastasis.

This was judged an advanced carcinoma of the breast with axillary metastasis and suitable for preoperative radiotherapy.

Therapy. Using 1 mm. of copper filtration, the patient was given 100 r daily to one, two, and sometimes three portals. The breast portals, with dimensions and dosage to each, were: lateral (10×15 cm.), 2,000 r; medial (10×15 cm.), 2,000 r; superior (8×10 and 6×8 cm.), 2,000 r; inferior (6×8 cm.), 2,000 r. All of these fields were directed tangentially to the chest wall, the breast being supported with sand bags so as to secure suitable apposition to the cones.

The axillary and supraclavicular structures were treated as a single unit. An anterior rectangular portal was centered about half way between the axillary fold and the base of the neck, with the long axis roughly parallel to the lateral border of the thorax. In this way all the struc-

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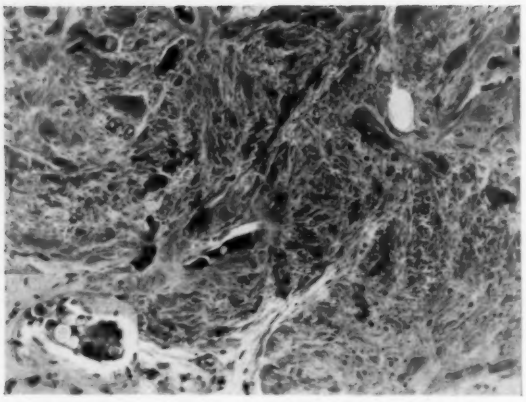

Fig. 1. Case 1. Tumor cells persisting in dense fibrotic stroma after irradiation, ×175, with an enlargement in the lower left corner showing cellular vacuolization, ×250.

tures were included, from 5 cm. above the clavicle to the base of the axilla, and from the suprasternal notch to the axillary fold. A similar field cross-fired this area posteriorly. With the patient lying in a position midway between supine and prone and on her right side with her arm raised over her head, a field was outlined covering the axilla, and the roentgen-ray beam was directed upward toward the base of the neck. The dosages to these fields were as follows: anterior clavicular (10×15 and 8×10 cm.), 2,200 r; posterior clavicular (10×15 and 8×10 cm.), 2,200 r; direct (10×10 and 8×10 cm.), 2,500 r. From May 6 through July 12, 8,000 r was given to the breast tumor, and 6,900 r to the axilla and supraclavicular regions.

Observation notes were recorded as follows: On May 20 the left breast was larger, with increased local temperature. On July 12 the tumor mass was difficult to measure. On September 9 the surgeon could not feel a tumor, and at this time a radical mastectomy was performed.

Gross Pathology (S.P. No. 68363). The measurements of the specimen were 27.3×20.8×4.5 cm., the breast, pectoral muscles, and axillary contents included. In the breast, 2.3 cm. beneath the skin, there was an ovoid, fairly well circumscribed, firm, grayish-white tumor. It measured 1.2×1×0.5 cm. Medially, and deeper in the breast, was another similar mass that measured 0.4×0.3×0.2 cm. Still another very small mass was palpated medially and cephalad to this second tumor.

Microscopic Pathology. Sections through all three of the tumor masses that were seen grossly showed persistent carcinoma. There were, however, evidences of marked radiation change. The tumor cells lay singly and in small groups (Fig. 1), scattered throughout a dense hyalinized connective tissue stroma. The nuclei were deeply hyperchromatic and varied greatly in size and shape. Some of them were gigantic and bizarre in shape, while others were very small. Many were pyknotic. None showed mitosis. The cytoplasms of many of the cells were poorly defined. They were eosinophilic, and often vacuolated (Fig. 1).

In the very dense stroma thickening of the walls of the blood vessels was seen.

In sections of the axillary nodes the carcinoma showed no radiation effect (Fig. 2). The cells were arranged in cords and atypical acini. Mitotic figures varied from one to six per high power field. Eight of the ten nodes sectioned contained tumor cells.

Case II (No. 475690). M. W., a colored woman, aged sixty-two, was admitted January 20, 1936, with a history of having noticed, for five months, that her right breast was larger than her left, and that in this enlarged breast there was a lump. Many years before, she recalled having had a right breast abscess treated by incision.

Local Examination. The right breast was enlarged and elevated, and the nipple deeply retracted. Directly posterior to the nipple was a hard tumor that measured 4×4.5 cm. The overlying skin was attached to it but showed no edema. The breast was not fixed to the chest wall. In the right axilla there were several moderately enlarged, firm, movable nodes. The tu-

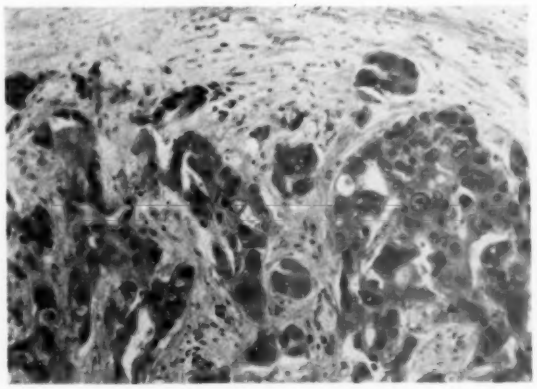

Fig. 2. Case 1. Carcinoma unaffected by irradiation in axillary lymph nodes. ×200.

mor being an advanced one, it was decided to give preoperative irradiation.

A biopsy was done on April 28, 1936, after the breast had been given 750 r. Sections showed a comparatively undifferentiated carcinoma. The tumor cells were arranged in cords and masses with very little tendency to form acini. The cells varied moderately in size and shape. There was an average of one mitotic figure per high power field.

Therapy. The filtration used in this case was 1 mm. of copper. One field per day was given 250 r. The breast was irradiated directly, from the medial and lateral sides, and from above. The direct field, 10×10 cm., later reduced to 6×8 cm., received 2,000 r. The lateral field, 10×10 cm., later reduced to 6×8 cm., received 2,250 r. The medial field, 10×10 cm., later reduced to 6×8 cm., received 2,250 r. The superior field, 6×8 cm., received 2,000 r. The lateral, medial, and superior fields were treated with the beam tangential to the chest wall.

The axillary fields, with dimensions and dosage to each were: anterior supraclavicular (6×8 cm.), 1,500 r; anterior axillary (10×10 cm.), 1,500 r; direct (6×8 cm.), 1,500 r. This direct axillary field was given in the same manner as in the previous case. From April 27 through September 2, 1936, 8,500 r was given to the breast tumor, and 4,500 r to the axillary and supraclavicular regions.

Observation notes were recorded as follows:

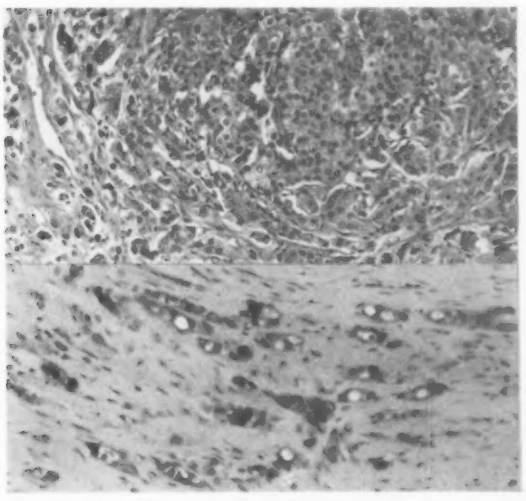

Fig. 3. Case 11. Above: well preserved carcinoma after irradiation. Below: irradiation effect shown by gigantic and pyknotic nuclei and by vacuoles in the cytoplasm. ×125.

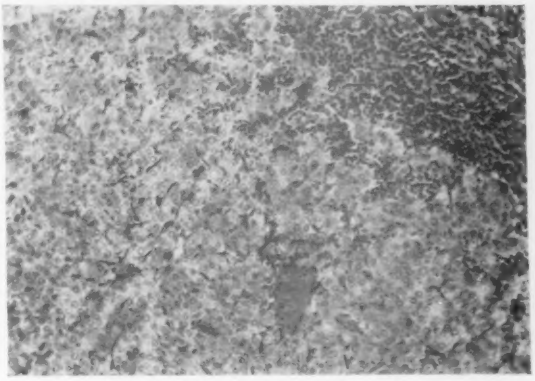

Fig. 4. Case 11. Axillary metastasis unaffected by irradiation. ×125.

On July 20, after twelve weeks of radiotherapy, a 4 cm. area of induration about the nipple remained. On August 19 the tumor measured 3×2 cm. No further reduction in the size of the tumor had occurred by November 2. A radical mastectomy was performed on November 5.

Gross Pathology (S.P. No. 62359). The specimen consisted of a female mammary gland with attached pectoral muscles and axillary tissues. The partially retracted nipple and surrounding skin presented a glistening surface, and in places the skin was scarred. On section there was, directly beneath the nipple, a mass of hard, white, encapsulated tumor that measured 1.5 cm. in diameter. It did not have the yellowish-white color so often seen in carcinoma.

Fourteen lymph nodes were found in the axillary tissues. The largest of these measured 1.5 cm. in diameter, and many of them apparently contained metastases.

Microscopic Pathology. Only scattered islands of carcinoma cells remained. Most of the tumor had been destroyed and replaced by dense, hyalinized connective tissue. The persisting cells were arranged in cords and small nests. In some of these the tumor cells appeared to be well preserved (Fig. 3). They varied from moderate to large in size, and were polyhedral and darkly eosinophilic. In other areas the persisting tumor cells showed marked evidence of radiation damage. Their nuclei varied much more in size and shape. Many were pyknotic. Some were gigantic. The cytoplasms often contained large vacuoles (Fig. 3). No mitoses were seen in any of the persisting tumor cells.

Five of the fourteen lymph nodes contained metastases. Here, the tumor cells did not appear

FIG. 5. Case III. Colloid type of carcinoma after irradiation. ×150.

to have been affected by the irradiation (Fig. 4). There was an average of one mitotic figure in each high power field.

CASE III. (No. 440370). M. L., a white woman, aged sixty, was admitted June 11, 1936. During a routine physical examination three years ago, a physician found a right breast tumor, and advised her to see a surgeon at once, but she procrastinated for three years.

Local Examination. The right breast was higher than the left. In the upper outer quadrant of the right breast there was a large, hard, nodular tumor that measured 10×8×6 cm. The skin over the tumor was fixed to it. There were two small, irregularly shaped, separate masses in the inframammary fold. These were fixed to the underlying chest wall and probably represented skin extensions. No enlarged axillary or supraclavicular lymph nodes were palpable.

The case was classed as an advanced one and preoperative irradiation was begun.

Therapy. With the usual factors and 2 mm. of copper filtration, 300 r was given daily to one of five portals. Two 6×8 cm. fields were used medially, one cephalad to the other. They received 2,100 r and 1,200 r, respectively. A single lateral portal was used. At first it was 10×10 cm. and later 6×8 cm.; 2,400 r was given through it. Another 2,400 r was given directly over the tumor through a 10×10 cm., and later a 6×8 cm. field. A dosage of 2,400 r was delivered to the inferior aspect of the breast. The beam was directed tangentially to the chest wall for all except the direct field. The axilla and supraclavicular regions were not treated.

Observation notes were recorded as follows: On August 15, twelve days after the last treatment, a severe desquamation with bleeding points was present, and there was marked reduction in the size of the tumor. Six weeks later, the main tumor measured 4×3 cm. and the small masses in the inframammary fold had disappeared. A radical mastectomy was done on October 3, 1936, soon after the skin reaction had cleared up.

Gross Pathology (S.P. No. 62067). The specimen consisted of a female mammary gland and attached skin, pectoral muscles, and axillary tissue. In the lower portion of the breast, somewhat lateral to the nipple, was a puckered area of skin. A section through this area revealed a tumor that measured 2.7×2.5×1.9 cm. located 2 cm. beneath the skin. In the tumor there were numerous small cavities containing blood or a bright yellow substance. There was present in another portion of the tumor an area of glistening gray tissue that resembled colloid carcinoma.

Eight lymph nodes were found in the axillary tissues. They varied from 0.2 to 1 cm. in diameter.

Microscopic Pathology. The tumor proved to be a colloid carcinoma. It was difficult to determine the extent of the radiation effect, but it was apparent at least that a large area of relatively unaltered colloid carcinoma persisted. Groups of comparatively regular tumor cells with small nuclei and acidophilic cytoplasm were scattered throughout large lakes of mucin (Fig. 5). In the fibrous portions of the tumor there was, however, definite evidence of radiation effect. There were masses of hyalinized collagen (Fig. 6), calcareous deposits, and some

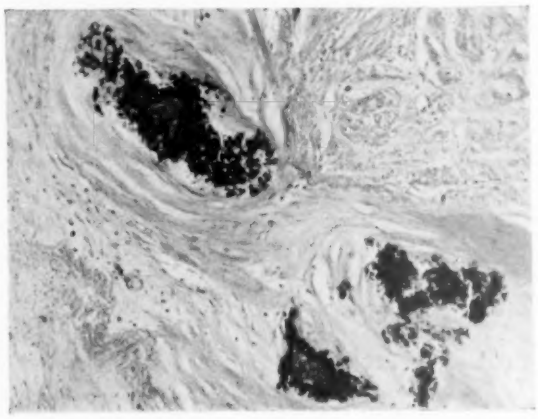

FIG. 6. Case III. Marked irradiation effect as shown by fibrosis and calcareous deposits. ×125.

areas of foam cells and cholesterol crystals. In these fibrous portions there remained groups of tumor cells that were arranged in atypical gland formation. The nuclei of some of these cells were pyknotic and of bizarre shapes and sizes. The cytoplasms were often vacuolated. No mitosis was seen.

All of the axillary lymph nodes were replaced by metastases. The tumor also infiltrated the axillary fat extensively. Mitotic figures averaged one in every high power field and no radiation effects were noted.

Case iv. (No. 422200). M. H., a colored woman, aged sixty-nine, was admitted September 3, 1935. Two weeks before, she had noticed a small lump in her right breast. There had been no noticeable increase in the size of the breast.

Local Examination. The breasts were equal in size and height. In the upper inner quadrant of the right breast there was a firm, irregularly shaped, but well defined tumor that measured about 2 cm. in diameter. It was situated over the third intercostal space just at the edge of the sternum. The tumor was fixed to the underlying tissues and the skin over the mass was attached to it. There was one small, not very firm node in the right axilla.

A biopsy was done on September 10, 1935. The sections showed a diffusely infiltrating carcinoma (Fig. 7). The tumor cells were arranged in slender cords and small masses. They did not form acini. They varied considerably in size and shape but tended to be small. They showed an average of two mitotic figures per high power field.

Therapy. With the usual factors, and using 2 mm. of copper filtration, the tumor was treated through a single direct portal. This was at first 8×10 cm. but was later reduced to 6×8 cm. Either 200 r or 250 r was given daily. The treatment started on September 13, and ended October 10, 1935, and during this period 5,000 r was administered. The axillary and supraclavicular regions were not treated.

Observation notes: On October 15, 1935, the skin was quite red and the tumor no longer palpable. Fourteen days later desquamation was present. By November 26, 1935, the radiodermatitis had entirely healed, and the tumor had completely disappeared as well as could be judged by careful palpation.

During the succeeding year the patient was examined frequently. In November, 1936, it

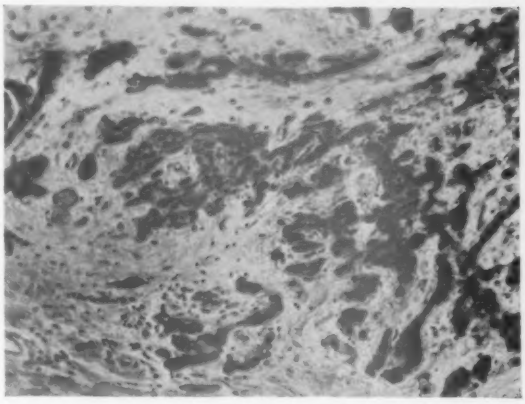

Fig. 7. Case iv. Biopsy taken before irradiation. ×125.

was noted that the node in the right axilla, which had been present since the treatment began, was enlarging. On November 20, 1936, a radical mastectomy was done. The excision included the removal of the chest wall down to the pleura at the site of the tumor. The second, third, and fourth costal cartilages were excised.

Gross Pathology (S.P. No. 62569). The specimen consisted of a female mammary gland with attached skin, pectoral muscles and axillary tissues, and three costal cartilages. On the skin, 8 cm. medial to the nipple, was a scarred area 6 cm. in diameter. Beneath it there was a cicatrix-like mass that measured 6×2×1 cm. It was not attached to the overlying skin or to the underlying muscle.

In the axillary contents there were four lymph nodes, varying from 0.5 to 1 cm. in diameter. Only the largest node appeared to contain metastases.

Microscopic Pathology. Sections from the irradiated area showed profound changes in all of the tissues. Only a few scattered nests of tumor cells remained (Fig. 8). These were similar in appearance and arrangement to those seen in the biopsy before irradiation and in the axillary nodes, although the irradiated cells were smaller. The stroma consisted of masses of dense collagen, much of which was markedly degenerated and almost acellular. There was practically no lymphocytic infiltration. Where not necrotic, the tissue contained many very small blood vessels with thin walls, but very few vessels of any size. The larger vessels all showed marked degeneration of the elastic lamina and intimal thickening. Many small veins contained organized thrombi.

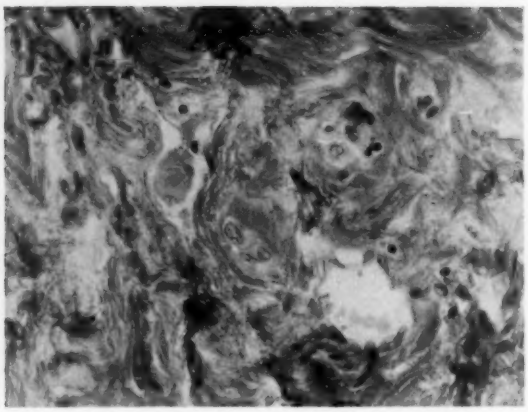

Fig. 8. Case iv. Persisting tumor cells scattered in dense hyalinized collagen. ×250.

The sections of the skin and the muscle also showed degenerative changes. These were most pronounced in the muscle tissue nearest the skin surface.

One axillary node was filled with metastatic carcinoma. The axilla did not receive any irradiation.

Case v. (No. 522147). F. M., a white woman, aged forty-seven, was admitted June 30, 1937, with a history of having been struck on the right breast one year before by a subway train door. Soon after the accident she noticed a tumor at the site of the injury, and about this same time bleeding from the nipple began. Both symptoms had persisted. During the first few months after discovery of the lump it did not appear to increase in size. It then began to enlarge. About this time a bluish discoloration appeared in the overlying skin, and this remained.

Local Examination. The right breast was larger and higher than the left. Beneath a dull blue area in the skin of the inferior outer quadrant there was a hard tumor that measured 10×9×6 cm. The central portion was fluctuant. Both nipple inversion and retraction were present. The tumor was attached to the skin and to the chest wall. In the right axilla there was a hard mass of axillary nodes 3×3 cm.

The case was regarded an inoperable one and it was decided to begin radiation treatment.

On July 20, 1937, a biopsy was done. The sections showed a papillary type of carcinoma (Fig. 9). The tumor cells were columnar and formed irregular papillary processes which often contained connective tissue cores. There was an average of one mitotic figure in each high power field.

Therapy. With the usual factors, and using 1 mm. of copper filtration, daily doses of 200 r were administered to one portal. The breast portals, with dimensions and dosage to each, were: lateral (8×10 cm.), 2,400 r; medial (10×15 cm. and later reduced to 8×10 cm.), 2,400 r; inferior (8×10 cm. and later reduced to 8×6 cm.), 2,400 r; direct (8×8 cm.), 600 r. The axillary portals, with dimensions and dosages, were: anterior supraclavicular (8×10 cm.), 2,000 r; anterior axillary (10×15 cm. and later reduced to 8×10 cm.), 2,000 r; posterior axillary (10×15 cm. and later reduced to 8×10 cm.), 2,600 r. In addition to the roentgen irradiation, a radium pack was applied to the region of the axillary metastases. The pack was composed of eight 12.5 mg. radium tubes placed so as to cover an area 4.5×5.5 cm. It was 3 cm. distant from the skin and delivered 10,125 mg-hr. over a period of eighteen days.

From July 20 through October 29, 1937, 7,800 r was given to the breast lesion, and 6,600 r to the axillary and supraclavicular regions, with the addition of 10,125 mg-hr. radium to the axilla.

Observation notes: There was a spontaneous rupture of the fluctuant area of the tumor on July 21, 1937. On August 18, the tumor was smaller, and on December 30, it measured 6.5×6.3 cm. On January 15, 1938, the surgeon who had followed the case since the beginning of treatment stated that although the primary tumor had decreased in size by one-half, the axillary metastases had not changed at all. It was therefore decided to do a radical mastectomy even though the hope of cure was very slight. Accordingly, on February 4, 1938, a radical mastectomy was performed.

Gross Pathology (S.P. No. 66316). The specimen consisted of a female mammary gland,

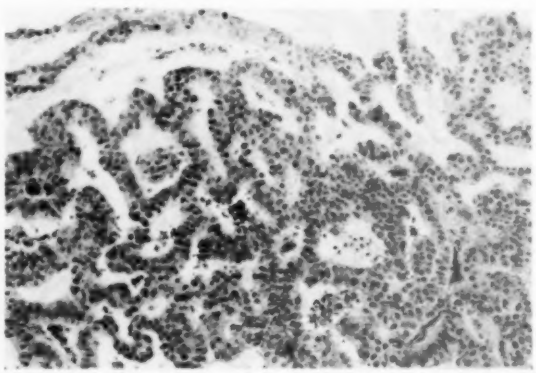

Fig. 9. Case v. Biopsy of papillary type of carcinoma before irradiation. ×125.

overlying skin, pectoral muscles and axillary tissues. It measured 30×23×4 cm. The tumor was situated in the lower central part of the breast. It measured 5×4×2.5 cm. It was soft, red, and partly cystic. A sinus tract led from it to the overlying skin.

Twenty-four lymph nodes were found in the axillary contents. All of them were small except one large, firm node that measured 2.5 cm. in diameter and appeared to contain metastases.

Microscopic Pathology. The sections showed that the irradiation had succeeded in destroying all of the breast tumor except a small zone a short distance beneath the skin over the central portion of the breast. This area was at the junction of the lateral and inferior fields and probably received considerably less irradiation than the remaining portion of the tumor. In this central zone the persisting tumor cells, although showing evidence of radiation damage in the form of vacuolization of the cytoplasm and gigantic and atypical nuclei, appeared to be well preserved and actively growing. This was proved by the presence of occasional mitotic figures (Fig. 10). Although most of these mitoses were atypical, some of them were comparatively regular.

The tumor had the same papillary structure that was noted in the original biopsy, but it was less evident because the papillary formations lay in a dense connective tissue stroma resulting from the heavy irradiation.

The severity of the radiation effect in the adjacent breast tissue was indicated by broad areas of degeneration in which there were deposits of hemosiderin, cholesterol crystals and calcium. Throughout most of the area originally occupied by the tumor, only hyalinized, atrophic tissue and areas of degeneration remained.

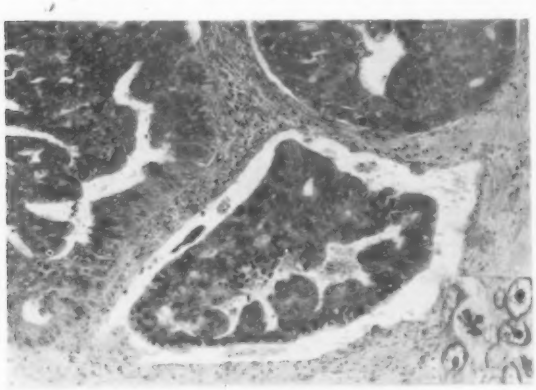

Fig. 10. Case v. A well preserved area of the tumor after irradiation, ×125. The inset shows persistence of mitosis, ×400.

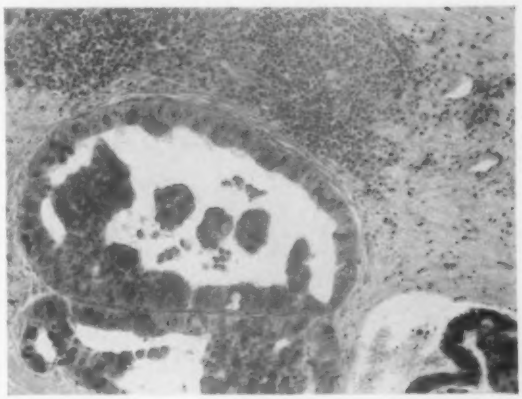

Fig. 11. Case v. Persistent carcinoma in an irradiated lymph node. ×125.

The large node which appeared to contain metastases in gross examination showed extensive hemorrhage and degeneration as a result of the irradiation which it had received. The lymphoid structure, which at one time must have been almost completely replaced by metastatic carcinoma, remained only as scattered foci of lymphocytes in the dense hyaline connective tissue around the periphery of the node. Several areas of carcinoma also persisted at the periphery of the node (Fig. 11). Here, the tumor cells retained their glandular and papillary arrangement. They closely resembled the tumor cells in the primary growth. They appeared to be actively growing, as indicated by the presence of occasional mitotic figures. Three of the twenty-three smaller nodes also contained metastases, and in them the metastatic tumor appeared even better preserved.

SUMMARY AND CONCLUSIONS

Detailed descriptions of five cases of breast carcinoma treated preoperatvely with roentgen rays have been presented. These cases were selected from a series treated in this manner, because the total dosages of radiation in them were as large as have been given in this hospital by the fractional method at 200 kv. Careful histological study of the operative specimens revealed persistence of the carcinoma in all of the cases, although the destructive effect of the radiation was clearly evident.

This finding has been the experience in all of the breast carcinomas removed after radiation treatment and studied in this laboratory.

THE EFFECT OF ROENTGEN THERAPY UPON INFECTIONS PRODUCED IN THE SKIN OF RABBITS WITH CULTURES OF *STREPTOCOCCUS HAEMOLYTICUS* AND *STAPHYLOCOCCUS AUREUS**

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THERE are numerous favorable reports of irradiation of various types of infections in man.[3,4,5,7,8,15] However, no one has adequately explained how the roentgen rays act upon the infection.

The experimental studies of roentgen therapy on infection were reviewed by Manges and Smith[9] in 1937. Many of the reported experimental studies do not concur with the usual clinical observations, and these authors have made the following objections to the experimental work: (1) that only small laboratory animals were used; (2) the irradiation was frequently general rather than local; (3) the amount of radiation was usually too great; (4) the irradiation was frequently given before the infection was produced.

Schaefer[12] studied the effect of irradiation on *Staphylococcus aureus* infections in the subcutaneous tissues of rabbits. Some of the 24 animals were irradiated with 450 r, and others received no treatment. He observed that necrosis appeared somewhat earlier in the irradiated animals, and the lesions healed in a shorter time. He studied the lesions from both groups histologically but noted no conspicuous differences in them.

Spinelli and Talia[14] injected guinea pigs subcutaneously with a virulent *Staphylococcus aureus* culture. They observed an earlier rupture of abscesses in the irradiated guinea pigs on the third to the fifth day whereas, in the non-irradiated pigs, the abscesses did not rupture until the thirteenth day. The effect of radiation therapy was about the same as that produced by surgical incision, and in most of their experiments they used about $\frac{1}{5}$ of the erythema dose. Cultures were also irradiated prior to inoculation, and the procedure apparently had no effect upon the subsequent course of the infection.

Pfalz[11] studied the effect of irradiation on mastitis produced with the hemolytic *Staphylococcus aureus* in guinea pigs. He concluded that dosages of 320 r had a beneficial effect on the course of the infection. Smaller and larger doses of roentgen rays did not have this effect.

Mischtschenko, *et al.*[10] made an extensive study of the effect of irradiation upon infections produced with *Staphylococcus aureus* in rabbits and guinea pigs. The effect of roentgen irradiation on diphtheria toxin injections in guinea pigs, as well as on pneumonia in mice, was also studied. They concluded that the quality of the roentgen ray, the dosage and the time exposure are important in the healing of infections. Eighty per cent of the skin erythema dose was found to be the optimal amount of roentgen rays necessary to inhibit an inflammatory reaction in rabbits. They also observed that large doses of roentgen rays can produce a spread of inflammation that may lead to septicemia.

Soto, Brunschwig and Schlutz[13] studied the effect of irradiation with 600 r on subcutaneous abscesses produced in rabbits by cultures of virulent colon bacilli. They concluded that such a dose of roentgen radiation reduced the severity of acute pyogenic infections in the skin and subcutaneous tissue of rabbits but did not necessarily hasten the healing of these lesions. A small number of irradiated lesions healed more slowly

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than the controls. They found that the optimum benefit of the roentgen rays was obtained when given within five hours after injection, and that irradiation became less effective as the suppurative phase of the infection became more prominent. Irradiation twenty-four hours prior to bacterial infection did not inhibit the severity of the subsequent lesions.

No one, so far as we can determine, has studied the effect of roentgen rays upon bacteria in vivo by quantitative, cultural methods. The following studies were undertaken to determine the effect of local irradiation upon relatively small skin abscesses produced by both avirulent and virulent hemolytic streptococci and also by *Staphylococcus aureus*. In most of the animals the number of bacteria in the skin lesions and the adjacent lymph nodes was determined when they were killed at different intervals after injection.

METHODS

A culture of *Streptococcus haemolyticus* (Strain H) was used in an avirulent and also in a highly virulent form. The avirulent culture will be designated as "stock." Using the method of Gay and Stone[6] the virulence was increased by repeated passage through the pleural cavity of rabbits, and the resulting culture will be designated as "passage." A culture of *Staphylococcus aureus* moderately virulent for rabbits was obtained from a furuncle.

The number of organisms injected into the skin was determined by special cultural methods.[1] An eighteen hour broth stock culture of *Streptococcus haemolyticus* contains about 600 million organisms per cubic centimeter. The amount injected was always 0.001 cc of culture so that about 600,000 organisms were the usual dose. The passage culture did not grow as heavily as the stock because of its lack of adaptation to growth on laboratory media so that an eighteen hour passage culture contained about 150 million organisms per cubic centimeter.

The *Staphylococcus aureus* culture contained about 800 million organisms per cubic centimeter. Because larger amounts of staphylococcus culture were required to produce a local abscess of size suitable for the study, the doses varied from 0.1 to 0.01 cc., or from 8 to 80 million organisms.

Healthy adult albino rabbits weighing from 2,000 to 2,400 gm. were used. The hair was removed with electric clippers. The animals were injected on both flanks about 4 cm. cephalad to the inguinal lymph nodes. The irradiation was usually given over the site of inoculation on the right side, that on the left side being used as a control. The skin lesions were observed and measured frequently. The average diameter, the height and the extent of necrosis are recorded in millimeters. For example, "20–5, nec. 8" indicates that the lesion was 20 mm. across, 5 mm. in height, with an area of necrosis measuring 8 mm. across. When the animals were killed, the skin lesion and the adjacent lymph nodes were removed and cultured to determine the number of viable organisms.[1] A culture of the heart's blood was made in every instance.

Irradiation. The lesions were irradiated through a portal sufficiently large to cover the entire lesion as well as a small part of surrounding normal skin. Unless otherwise specified, irradiation was delivered at 100 kv. from a distance of 25 cm.

EXPERIMENT I

To determine the effect of irradiation prior to injection, 4 animals were irradiated over the skin with 100 r, 2 of them twenty-four hours before, and 2 immediately before injection. Each animal was then injected with 0.001 cc. of *Streptococcus haemolyticus* culture, 2 receiving stock, and 2 passage culture. They were killed about five hours later. The results of the cultural studies made on both skin and lymph nodes are shown in Table 1. In 1 of 2 animals that received the stock culture the number of organisms recovered from the non-irradiated lesion was greater, and in the other that from the irradiated lesion was greater, the average number from irradiated and non-irradiated lesions being approximately the same. More organisms were recovered from the skin of the animals injected with passage culture, and in each instance about five times as many organisms were recovered from the irradiated site.

EXPERIMENT II

Seven rabbits each received two intradermal injections of 0.001 cc. of stock culture of *Streptococcus haemolyticus*, and one of the lesions on

TABLE I

EFFECT OF IRRADIATION PRIOR TO INTRADERMAL INJECTION WITH 0.001 CC. OF STOCK OR PASSAGE CULTURE OF HEMOLYTIC STREPTOCOCCUS

Rabbit No.	Irradiation: Interval before Injection	Irradiation: Dose of Roentgen Rays	Culture	Interval between Irradiation and Death	Number of Organisms Recovered: Skin	Number of Organisms Recovered: Lymph Nodes
1	—	—	Stock	5½ hr.	55,000	240
	24 hr.	100 r	Stock		15,000	0
2	—	—	Stock	5½ hr.	15,000	0
	Immediately	100 r	Stock		65,000	60
3	—	—	Passage	5 hr.	100,000	110
	24 hr.	100 r	Passage		500,000	840
4	—	—	Passage	5 hr.	1,400,000	0
	Immediately	100 r	Passage		10,100,000	0

each animal received a single treatment that varied from 50 to 400 r, at intervals of from one to three days after injection (Table II). The lesions were measured daily during one week and then the animals were killed. In every instance the irradiated lesion was larger than the non-irradiated. Necrosis developed in the lesions of 3 rabbits; it appeared somewhat earlier and was larger in the treated lesions. In 5 instances the lesions were cultured, and more organisms were recovered from the irradiated lesion in every case. In only one instance were organisms recovered from the inguinal lymph node, and this was on the side of and adjacent to an irradiated site. Doses of 400 r apparently did not influence the course of infection any more than 50 r.

EXPERIMENT III

To determine the effect of irradiation on skin lesions produced with a virulent hemolytic streptococcus, each of 5 rabbits received two intradermal injections of 0.001 cc. of passage culture, and were then given doses of roentgen rays that varied from 50 to 400 r at intervals of from twelve hours to three days after injection

TABLE II

EFFECT OF IRRADIATION ON SKIN LESIONS IN RABBITS INJECTED INTRADERMALLY WITH 0.001 CC. (600,000 ORGANISMS) OF AN AVIRULENT HEMOLYTIC STREPTOCOCCUS

Rabbit No.	Amount of Injection	Irradiation: Dose of Roentgen Rays	Irradiation: Interval after Injection	Size of Lesion at Time of Irradiation	Interval between Irradiation and Death	Size of Lesion	No. of Organisms Recovered: Skin	No. of Organisms Recovered: Lymph Nodes
1	0.001 cc.	—	—	20–3	1 week	26–5 nec. 6	—	—
	0.001 cc.	50 r	2 days	51–3		35–5 nec. 10	—	—
2	0.001 cc.	—	—	14–3	1 week	16–3 nec. 5	10,000,000	0
	0.001 cc.	100 r	1 day	14–3		19–5 nec. 7	19,200,000	0
3	0.001 cc.	—	—	22–4 nec. 2	1 week	31–5 nec. 7	152,000,000	0
	0.001 cc.	100 r	3 days	28–4 nec. 4		33–4 nec. 7	197,000,000	0
4	0.001 cc.	—	—	8–2	1 week	10–1	0	0
	0.001 cc.	100 r	3 days	12–3		11–2	945,000	0
5	0.001 cc.	—	—	7–2	1 week	7–2	15,000	0
	0.001 cc.	100 r	3 days	8–3		8–2	45,000	0
6	0.001 cc.	—	—	20–3	1 week	18–5	360,000	0
	0.001 cc.	200 r	2 days	27–3		39–5	5,000,000	26
7	0.001 cc.	—	—	17–3	1 week	16–4	—	—
	0.001 cc.	400 r	2 days	23–3		23–6	—	—

TABLE III

EFFECT OF IRRADIATION ON SKIN LESIONS IN RABBITS INJECTED INTRADERMALLY WITH 0.001 CC. (150,000 ORGANISMS) OF A VIRULENT HEMOLYTIC STREPTOCOCCUS

Rabbit No.	Amount of Injection	Irradiation: Dose of Roentgen Rays	Irradiation: Interval after Injection	Size of Lesion at Time of Irradiation	Interval between Irradiation and Death	Size of Lesion	No. of Organisms Recovered: Skin	No. of Organisms Recovered: Lymph Nodes
1	0.001 cc.	—	—	27–5 nec. 9	7 days	30–5 nec. 8	6,400,000	—
	0.001 cc.	50 r	2 days	24–5 nec. 4		33–6 nec. 5	2,400,000	—
2	0.001 cc.	—	—	—	5 days	18–3 nec. 5	40,800,000	0
	0.001 cc.	100 r	12 hr.	—		25–5 nec. 5	55,300,000	500
3	0.001 cc.	—	—	13–3	5 days	7–1 nec. 3	720,000	0
	0.001 cc.	100 r	1 day	15–3		10–3 nec. 3	100,000	0
4	0.001 cc.	—	—	24–5 nec. 3	9 days	20–4 nec. 4	36,000,000	0
	0.001 cc.	100 r	3 days	20–5 nec. 2		15–4 nec. 4	17,900,000	0
5	0.001 cc.	—	—	17–5 nec. 4	9 days	11–4 nec. 2	2,700,000	0
	0.001 cc.	100 r	3 days	17–5 nec. 7		17–5 nec. 4	700,000	0
6	0.001 cc.	—	—	19–5 nec. 7	6 days	24–7 nec. 6	15,400,000	—
	0.001 cc.	200 r	2 days	23–5 nec. 9		36–10 nec. 7	3,800,000	—
7	0.001 cc.	—	—	18–5 nec. 8	6 days	23–5 nec. 4	9,000,000	—
	0.001 cc.	400 r	2 days	25–5 nec. 5		22–7 nec. 2	2,500,000	—

TABLE IV

EFFECT OF IRRADIATION ON SKIN LESIONS IN RABBITS IRRADIATED 4½ HOURS AFTER AN INTRADERMAL INJECTION OF 0.001 CC. (150,000 ORGANISMS) OF A VIRULENT HEMOLYTIC STREPTOCOCCUS

Rabbit No.	Dose of Roentgen Rays	Size of Lesion 24 Hours after Irradiation	Interval between Irradiation and Death	Size of Lesion	Number of Organisms Recovered: Skin	Number of Organisms Recovered: Lymph Nodes
1	—	18–4	1 day	18–4	7,500,000	9,500
	600 r	20–4		20–4	19,700,000	14,640
2	—	21–4 nec. 3	2 days	39–5 nec. 3	10,200,000	0
	600 r	22–4 nec. 3		48–6 nec. 7	30,500,000	4,370
3	—	21–4 nec. 6	2 days	25–6 nec. 7	4,750,000	1,560
	600 r	27–3 nec. 6		30–6 nec. 9	4,500,000	2,900
4	—	18–4	3 days	35–5	3,100,000	0
	600 r	18–4 nec. 3		53–6 nec. 6	59,000,000	60
5	—	23–4 nec. 3	3 days	28–5 nec. 4	1,100,000	0
	600 r	18–4		36–6 nec. 5	9,100,000	20
6	—	19–4	18 days	20–2 nec. 8	—	—
	600 r	15–3		14–2 nec. 2	—	—
7	—	18–4	18 days	16–2 nec. 5	—	—
	600 r	18–3		21–2 nec. 13	—	—
8	—	13–2	18 days	14–5	—	—
	600 r	15–4 nec. 3		16–2 nec. 6	—	—
9	—	19–4 nec. 5	29 days	10–2 nec. 4	—	—
	600 r	18–4 nec. 5		14–3 nec. 7	—	—
10	—	17–4 nec. 4	29 days	7–1	—	—
	600 r	19–4 nec. 5		13–4 nec. 4	—	—

TABLE V

EFFECT OF IRRADIATION ON SKIN LESIONS IN RABBITS IRRADIATED 24 HOURS AFTER AN INTRADERMAL INJECTION OF STAPHYLOCOCCI

Rabbit No.	Amount of Injection	Dose of Roentgen Rays	Size of Lesion at Time of Irradiation	Size of Lesion 48 Hours after Irradiation	Interval between Irradiation and Death	Size of Lesion	No. of Organisms Recovered	
							Skin	Lymph Nodes
1	0.1 cc.	—	43–2 nec. 3	25–3 nec. 2	3 days	21–4 nec. 2	42,000,000	—
	0.1 cc.	100 r	40–2 nec. 2	18–3 nec. 3		19–4 nec. 2	17,000,000	—
	0.01 cc.	—	12–1	3		3	4,000	—
	0.01 cc.	100 r	14–1 nec. 1	5–1 nec. 2		5–1 nec. 2	169,000	—
2	0.1 cc.	—	38–2 nec. 1	23–3 nec. 3	8 days	16–4 nec. 6	25,000,000	0
	0.1 cc.	100 r	35–2 nec. 2	44–3 nec. 3		32–4 nec. 6	300,000	0
	0.01 cc.	—	23–2	20–1 nec. 2		4–2	0	0
	0.01 cc.	100 r	23–2	14–.5 nec. 2		4–2	13,500	0
3	0.05 cc.	—	23–2 nec. 3	23–3 nec. 4	6 days	14–5 nec. 5	7,000,000	0
	0.05 cc.	100 r	14–3 nec. 2	17–3 nec. 4		14–5 nec. 5	1,800,000	0
	0.01 cc.	—	6–1 nec. 2	12–2 nec. 2		8–3 nec. 3	26,000	—
	0.01 cc.	100 r	5–1	6–1		5–1	2,000	—
4	0.05 cc.	—	35	34–4 nec. 2	7 days	11–2 nec. 2	—	—
	0.05 cc.	100 r	41–2	40–5 nec. 2		12–2 nec. 2	—	—
	0.01 cc.	—	19–1	23		8	—	—
	0.01 cc.	100 r	19–1	17–2		14	—	—
5	0.05 cc.	—	18	20–3 nec. 2	9 days	12–2 nec. 6	10,000	0
	0.05 cc.	100 r	18–2	27–4 nec. 2		13–3 nec. 7	1,890,000	0
	0.01 cc.	—	13	12–1 nec. 1		neg.	—	—
	0.01 cc.	100 r	12	10–1		neg.	—	—
6	0.05 cc.	—	19–3 nec. 5	19–4 nec. 5	18 days	18–3	—	—
	0.05	100 r	21–3 nec. 5	20–4 nec. 4		25–4	—	—
	0.01 cc.	—	6–1 nec. 1	6–2		4–1	—	—
	0.01 cc.	100 r	6–1 nec. 1	6–1		neg.	—	—

(Table III). The animals were permitted to live from five to nine days, and the lesions were observed and measured daily. In 5 instances the irradiated lesions were larger than the non-irradiated at the time the animals were killed. There was no conspicuous difference in the extent of necrosis in the treated and non-treated lesions except that it appeared somewhat earlier in the former. There was also a fairly extensive erythema in the treated lesions that usually persisted for about forty-eight hours after injection. The skin lesions were cultured in every instance. A larger number of organisms was recovered from the non-treated lesions of these animals with the exception of that from Rabbit No. 2. A larger number of streptococci was recovered from the irradiated lesion of this animal than from any other lesion in this group. Five hundred organisms were recovered from the lymph node adjacent to the irradiated lesion, and streptococci were also grown from the heart's blood.

Two rabbits not included in Table III were infected in a similar manner, and one lesion was treated with 100 r; they were observed for thirty and forty-three days respectively, or until the lesions were completely healed. No difference was noted in the rate of healing of the treated and non-treated lesions.

EXPERIMENT IV

Because of the recent publication of Soto, Brunschwig and Schlutz[13] stating that 600 r, when given within five hours of injection, has some inhibiting effect upon *B. coli* infections, it seemed advisable to determine the effect of such irradiation on streptococcus infections.

Ten rabbits were injected at two sites with 0.001 cc. of passage culture, and one lesion of each animal was irradiated with 600 r, four and a half hours after injection (Table IV). In this experiment the deep roentgen radiation was delivered at 200 kv. from a distance of 50 cm. and filtered through 1 mm. of copper. After

TABLE VI

EFFECT OF REPEATED IRRADIATION ON SKIN LESIONS IN RABBITS 24 AND 48 HOURS AFTER AN INTRADERMAL INJECTION OF STAPHYLOCOCCI

Rabbit No.	Amount of Injection	Dose of Roentgen Rays		Interval between Last Irradiation and Death	Size of Lesion	Number of Organisms Recovered	
		24 hr.	48 hr.			Skin	Lymph Nodes
1	0.01 cc.	—	—	2 days	15–1	6,200	—
	0.01 cc.	—	200 r		17–1	100	—
	0.01 cc.	200 r	400 r		17–1	0	—
	0.02 cc.	—	—		14–1	5,800	0
	0.02 cc.	—	200 r		16–1	2,600	0
	0.02 cc.	200 r	400 r		15–1	500	0
2	0.01 cc.	—	—	3 days	16	—	0
	0.01 cc.	100 r	100 r		neg.	—	0
	0.02 cc.	—	—		7	0	0
	0.02 cc.	100 r	100 r		0	0	0
3	0.01 cc.	—	—	4 days	neg.	—	—
	0.01 cc.	100 r	100 r		neg.	—	—
	0.02 cc.	—	—		neg.	—	—
	0.02 cc.	100 r	100 r		neg.	—	—

twenty-four hours the irradiated lesions in 6 of the rabbits were larger than the non-irradiated, and there was considerably more erythema and earlier necrosis. When the animals were killed at intervals of from one to twenty-nine days after injection, 9 of the irradiated lesions were larger, and 8 showed more necrosis. The skin lesions of 5 animals were cultured, and in 4, more organisms were recovered from the irradiated lesion. Streptococci were recovered from the lymph node draining the irradiated lesion in all instances, whereas they were recovered from only 2 of 5 nodes adjacent to non-treated lesions. Five of the animals were permitted to live, and the lesions observed over a longer period of time. Although there was no conspicuous difference in size between the treated and non-treated sites of infection after eighteen and twenty-nine days observation, the irradiated lesions were somewhat larger.

TABLE VII

EFFECT OF ROENTGEN RADIATION FILTERED THROUGH 1 MM. OF ALUMINUM ON SKIN LESIONS OF RABBITS 24 HOURS AFTER AN INTRADERMAL INJECTION OF STAPHYLOCOCCI

Rabbit No.	Amount of Injection	Dose of Roentgen Rays	Interval between Irradiation and Death	Size of Lesion	Number of Organisms Recovered	
					Skin	Lymph Nodes
1	0.02 cc.	—	3 days	18	0	—
	0.02 cc.	200 r		18	3,500	—
	0.02 cc.	100 r		5–2	550,000	—
	0.05 cc.	—		7–2	430,000	—
	0.05 cc.	200 r		7–2	53,000	—
	0.05 cc.	100 r		10–1	550,000	—
2	0.05 cc.	—	4 days	3–1	800	180
	0.05 cc.	200 r		15–5	130,000	0
	0.05 cc.	—		9–3	13,000	—
	0.05 cc.	100 r		14–4	25,000	—

EXPERIMENT V

To determine the effect of irradiation on staphylococcus infections, 12 rabbits were injected intradermally at two sites with either 0.1, 0.05 or 0.01 cc. of a *Staphylococcus aureus* culture (Table v), and twenty-four hours later one of the lesions received a single treatment with 100 r. After forty-eight hours the treated and non-treated lesions were similar in size. The lesions were observed for intervals varying from three to eighteen days, and no difference in size or character was observed. There was no consistent difference in the number of organisms recovered from the treated and non-treated lesions. In no instance were organisms recovered from the lymph nodes.

EXPERIMENT VI

Experiments were also done to determine the effect of repeated irradiation on eight sites of injection produced with staphylococcus in the skin of 3 rabbits (Table vi). Four of the lesions received 100 r at intervals of twenty-four and forty-eight hours after injection. No conspicuous differences were observed in the treated as contrasted with the non-treated lesions. Another animal (Rabbit No. 1, Table vi) received larger doses of roentgen radiation and was killed forty-eight hours after the last treatment. More organisms were recovered from the non-treated lesion; however, there was no difference in size at this time. Two more rabbits (Nos. 1 and 2, Table vii) were injected with similar amounts of the same culture and treated with various doses of roentgen radiation filtered through 1 mm. of aluminum. In most instances more bacteria were recovered from treated than from non-treated lesions.

DISCUSSION

We have endeavored to meet the objections of Manges and Smith[9] to earlier experimental work. In the experiments reported, the radiation has been applied locally in small doses after the establishment of an infection. In other words, the usual methods for treatment of superficial infections in man have been employed. To offset the individual variation of animals, both control and treated lesions have been established in the same animal.

Although there are differences in the size of the cutaneous lesions, as well as in the number of organisms recovered, they are not great, and in several instances may be considered within the limits of error of the method used. To determine whether such differences are of statistical significance, it would be necessary to carry on many more experiments than have been reported here.

It was observed that necrosis usually appeared from one to two days earlier in the irradiated than in the non-irradiated lesions. It was usually associated with considerable erythema that lasted for about forty-eight hours after treatment. This earlier development of necrosis following irradiation is in accord with the observations of others.[12,14] When necrosis appears there is usually an escape of purulent exudate followed by fairly rapid diminution in the size of the lesion. This observation may explain the reports of earlier healing of irradiated lesions in man.

When deep roentgen therapy was given in doses of 600 r a few hours after the establishment of streptococcus infections, it was found to have an unfavorable effect upon the course of the treated lesions since they were larger and contained more organisms than controls. This observation is not in accord with the report[13] that a similar type of irradiation produced an earlier healing of colon bacillus infections in rabbits.

The inguinal lymph nodes adjacent to 18 irradiated and 18 control lesions produced by *Streptococcus haemolyticus* were cultured at various intervals after injection. Organisms were isolated from 12 of the 36 lymph nodes; they were present in 9 of the nodes on the irradiated side and in only 3 on the control side. The number of organisms recovered from the irradiated nodes was greater in all except one instance. Streptococci would probably have been recovered from a larger number of these lymph nodes had all the animals been killed within seven days after injection.[2] It is of interest that organisms were recovered from only 3 of 14 inguinal nodes of animals infected with stock culture whereas they were isolated from 10 of 22 nodes of rabbits receiving the passage culture. Thirteen inguinal lymph nodes draining staphylococcus infections were

cultured; organisms were recovered from 1 lymph node only, and this was on the non-treated side. These experiments demonstrate very well the high degree of invasion of virulent as compared with avirulent streptococci, and also that the staphylococcus is practically non-invasive.

SUMMARY AND CONCLUSIONS

1. Irradiation of the skin before infection had no evident effect upon the course of hemolytic streptococcus infections.

2. In animals irradiated after infection, necrosis developed earlier in treated than non-treated lesions.

3. Of 50 irradiated streptococcus and staphylococcus skin infections, 32 were larger than the non-irradiated controls, 9 were smaller and 9 of about equal size at the time when the animals were killed.

4. Larger numbers of organisms were recovered from 17 of the 30 irradiated lesions that were cultured, and from 13 of their controls.

5. The number of organisms recovered from the lymph nodes adjacent to the irradiated site was usually greater than from those on the untreated side.

6. The effect of roentgen treatment upon experimental skin infections was to increase the size of the lesions, to produce more necrosis and enhance the invasiveness of the microorganisms.

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A PRACTICAL ROENTGENOGRAPHIC DENSITOMETER

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IN THE child development studies made by the late Dr. T. Wingate Todd, and in the work of nutritionists and allergists, attempts have been made to determine

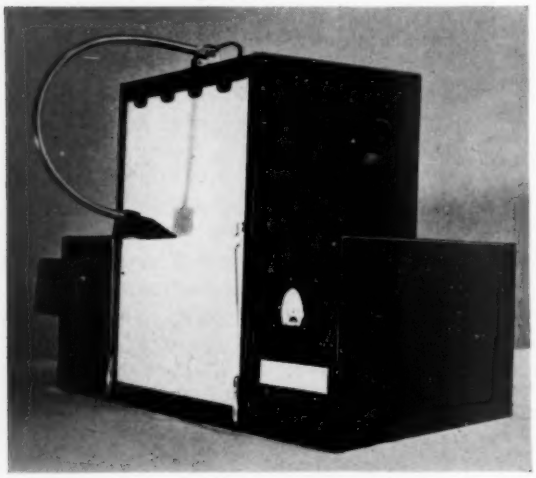

FIG. 1. The roentgenographic densitometer.

density of muscle, bone and other tissues by means of roentgenographic shadows. In most of this work densities were determined visually and hence personal factors introduced variabilities which seriously affected the value of the studies. A search for a practical objective method for making these measurements resulted in the design and construction of the instrument described here.

It is well known in physics that the absorption of roentgen rays is very nearly proportional to the mass of the material penetrated. By maintaining the entering roentgen-ray beam at a constant intensity and knowing the thickness of the material it traverses, it is possible to gain some idea of the density of that material by measuring the intensity of the emerging beam of roentgen rays. In roentgenograms, taken under normally controlled conditions, we have permanent intensity records of these emerging beams.

To measure such intensities a film is placed on a spot of light of constant intensity and a photo-cell and galvanometer are used to register the amount of light which traverses the film. The instrument is calibrated in tissue density units and the proper technique followed to automatically eliminate personal errors and the variables due to film exposure and development. An easily read steady-deflection galvanometer and a calibration table giving the relative tissue density for each millimeter of deflection reduce the operator's duties to a minimum.

The complete instrument requires 110 volt alternating current only and is quite portable, all parts being enclosed in a single metal case as shown in Figure 1. Since it is frequently necessary to view a film as a whole, the large central portion includes a special type roentgenogram illuminator 16 inches wide and 18 inches high.[1] Cold blue light for the flashed opal glass of the illuminator is supplied by two 18-inch tubular fluorescent bulbs[2] mounted in a shallow reflector. Four soft rubber rolls at the top and two adjustable 10-inch rubber-tipped arms extending upward from the bottom corners hold the film on the glass at any desired angle. An aluminum cone (Fig. 2) projects through the center of the illuminator reflector to conduct light from a 150 watt bulb[3] in the rear which supplies a densitometer spot for the center of the glass. In order to hold this spot at constant intensity a voltage stabilizer[4] in the 110

[1] Picker X-ray Company "Picker Fluorescent Illuminator." (All superscript numbers also indicate respective parts in Fig. 2.)

[2] General Electric Mazda 15 watt, Blue Fluorescent.

[3] General Electric Mazda 150 watt internally silvered parabolic "Reflector."

[4] Raytheon Voltage Regulator, 250 watt, 115 volt.

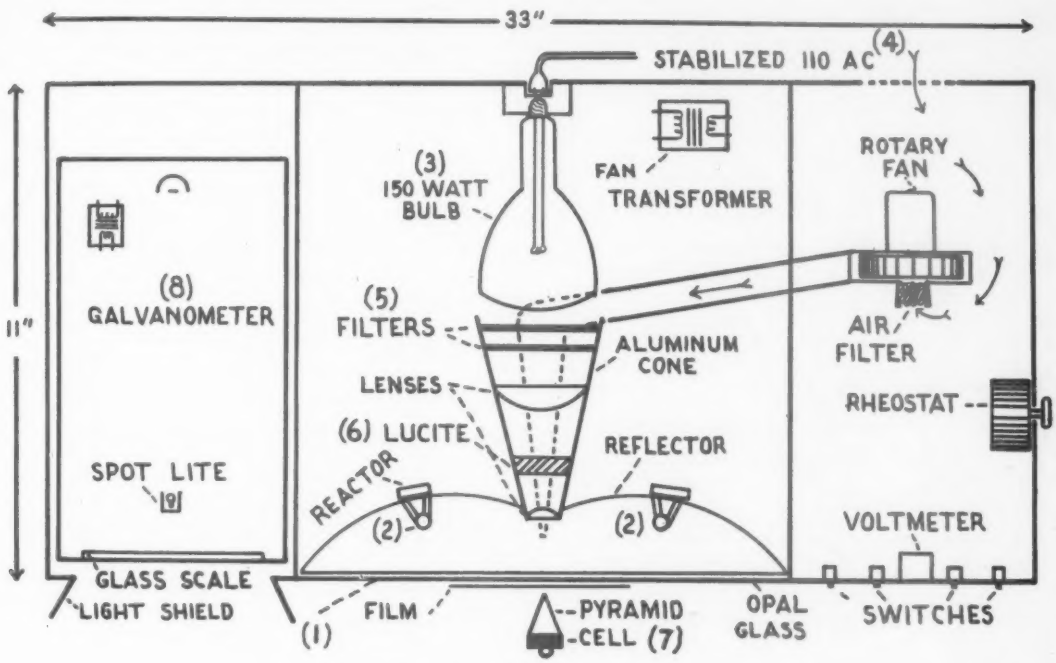

FIG. 2. Top-view, cross sectional diagram.

volt alternating current supply is necessary. Since the heat generated by the bulb must not cause a temperature rise of more than 10° C. above room temperature to prevent film damage, a small rotary fan located in the right-hand compartment is employed to cool the bulb and optical cone. A transformer supplying this fan permits an adjustment of speed, and a pad of glass wool in the intake filters the cooling air.

The optical cone contains two solid glass filters,[5] the first of which removes all wave lengths of light above 54 millimicrons and the second all below 50 millimicrons. These filters serve the dual purpose of removing heat from the beam and of transmitting only green wave lengths which have the most effect upon the photo-electric cell. Monochromatic light is desirable because films may exhibit a selective absorption of wave lengths which would introduce a variable not due to density. After traversing these filters the beam of green light passes through a 4-inch condensing lens, a 3-inch disc of lucite[6] which removes more heat, and finally a 2-inch condensing lens. This system projects a cool green beam on the back of the flashed-opal glass and the glass in turn disperses the beam so that a spot of uniform intensity about 1 inch in diameter is formed as a background for the film. As stated above, films can be placed and held in any position upon this spot.

A photo-electric cell[7] for scanning any square millimeter of film is mounted on a hinged, semicircular copper tube which can be turned out of the way while the illuminator is being used for other purposes. The active cell surface is covered by a 2-inch brass pyramid which has a 1 mm. aperture at its apex. Heavy chromium plating on the interior surface of this hollow pyramid prevents excessive absorption of the small amount of light entering the aperture, reflecting practically all of it upon the active cell surface. Without this reflective coating the sensitivity of the instrument is decreased by as much as 50 per cent.

Minute differences of potential generated by light acting on the cell are led by shielded wires through the supporting tube

[5] Corning 4" circular filters, Nos. 352 and 452.
[6] DuPont Methyl Methacrylate rod stock.
[7] General Electric "Light-Sensitive" cell.

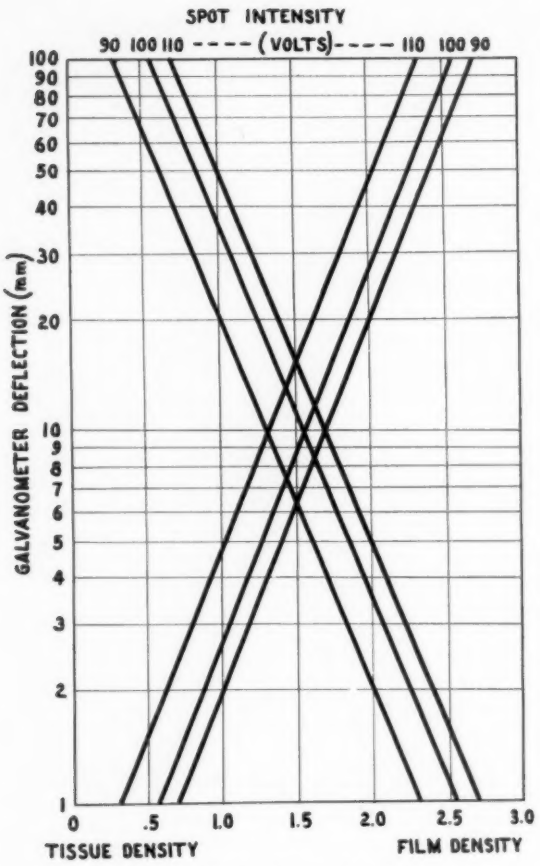

Fig. 3. Logarithmic tissue density and film density curves.

to a sensitive galvanometer in the left hand compartment. This is a portable galvanometer[8] with an enclosed, reflected-beam spot that is read on a 100 mm. ground glass scale. A light shield placed around the scale permits readings to be taken in a normally lighted room. Both galvanometer and cooling fan are mounted on sponge rubber cushions to eliminate vibration.

A voltmeter, with full scale calibration of 90 to 130 volts, and a rheostat are connected in the 150 watt bulb circuit so that the intensity of the spot can be varied in known steps.

The rheostat control projects from the right-hand side of the illuminator cabinet and four switches and the voltmeter are conveniently placed on the right-hand front panel. From left to right, switch number one connects the cell to galvanometer; number two activates a small transformer in the galvanometer case which supplies the enclosed spotlite for the scale; number three simultaneously turns on the green densitometer spot and rotary fan so that cooling starts before excessive heating occurs; number four controls the blue viewing lights which are turned off when density readings are being made. From thirty seconds to one minute after the fan and green spot are turned on the changes due to heating and cell fatigue become negligible and readings can be taken.

Calibration curves made by moving an Eastman neutral tint wedge across under the millimeter aperture and plotting density against millimeters of galvanometer deflection are shown above "film density" in Figure 3. Three logarithmic curves were obtained for three spot intensities and repeated tests on these settings gave the same curves with variations of less than ± 1 per cent. These three intensities were chosen so that the entire range of film densities found in practice would be included.

Since film densities are inversely related to tissue densities (light portions of film represent shadows of dense tissue) a method was sought to obtain tissue density values which increased as the galvanometer deflection values increased. For the present it has been decided to use film density units for relative tissue densities until a calibration study can be completed which will permit their conversion into true physical tissue density units. To obtain these more intelligible units the curves were again plotted on semi-logarithmic paper and their slopes reversed by plotting the same abscissa values for film densities in reverse order (Fig. 3 above "tissue density"). By this means relative tissue density values can be found which increase as the galvanometer deflections increase and the values obtained in this manner bear a true relation to actual tissue densities although they are not yet on an absolute basis. To simplify the operator's duties a table was compiled

[8] Rubicon Company No. 3403H Portable Spot-lite Galvanometer.

TABLE I

ABBREVIATED CALIBRATION TABLE FOR RELATIVE TISSUE DENSITIES CORRESPONDING TO EACH MILLIMETER OF GALVANOMETER DEFLECTION

Galvanometer Deflection mm.	Relative Tissue Density (based on film density units)				
	Spot Intensity 90 volts		Spot Intensity 100 volts		Spot Intensity 110 volts
1	.322	Conversion	.522	Conversion	.699
10	.363	Factor	.592	Factor	.740
20	.414		.643		.791
30	.471		.700		.848
40	.567	+ →	.766	+ →	.914
50	.615	.229	.844	.147	.992
60	.709	← −	.939	← −	1.086
70	.831		1.060		1.208
80	1.000		1.229		1.377
90	1.281		1.510		1.658
100	2.322		2.551		2.699

which gives there lative tissue density value for each millimeter of galvanometer deflection at each of the three spot intensities. Table 1 gives an abbreviation of this table. In case a change in intensity is necessary to measure excessively dense or light spots a single factor is added to or subtracted from these values to convert from one column to the next.

In spite of standardized exposure and developing techniques, variations do occur in the over-all density of roentgenograms taken at different times. Several years ago an aluminum "densimeter" was devised and standardized by the Brush Foundation for use as a guide in visual comparison of roentgenograms. This consists of ten lengths of the same gauge sheet aluminum, riveted together to form steps about 1 cm. square. When the roentgenogram is being taken this is placed on a portion of the film where it will not interfere with the shadow of the object being roentgenographed. Upon exposure and development, the "densimeter" shadow consists of small squares which vary in density from dark to light in successive steps. When two roentgenograms are compared visually these ladders are placed in juxtaposition on the illuminator and moved up and down until two adjoining squares appear to have the same density. By noting the relative positions of these two squares in their respective ladders, an estimate of the difference in exposure and development can be made and visual comparison of the roentgenograms done with this in mind.

A similar procedure is followed in using the densitometer. One reading is taken on a given square of each film in order to establish basic differences in films that are not due to the objects being roentgenographed. These basic values, usually small, are added to or subtracted from each other density value taken from their respective films, depending upon whether they are larger or smaller than the average film value which has been selected as standard. In films without a ladder this basic measurement can be made on a corner where there is no shadow of the object being roentgenographed.

Variations in density over a single film resulting from the radial dispersion of roentgen rays from the tube can easily be observed by means of the densitometer. It is possible to tell whether or not an exposed but blank film was properly centered by

comparing readings taken along two perpendicular lines which intersect at the film's center. Such variations are always small, however, and if future work demands extreme accuracy these corrections can be made.

Two studies are now in progress in this laboratory which will establish actual physical tissue density values to replace the relative values now based on film densities. In the first study roentgenograms are being taken of fluids of known densities and depths and their resultant shadows measured by the densitometer. The second method requires roentgenograms of the same animal bones in three conditions: first in the body, secondly when cleaned but wet, and thirdly when baked dry. These bones are then ashed for a chemical analysis of mineral content and all of these values correlated with densitometric readings of their respective films.

Thus the instrument as it now stands can be used to show individual tissue density differences and relative changes in density over a period of time. Each density determination requires only a few seconds, and the operator's duties do not require technical training or laboratory conditions. Upon completion of the calibration study the relative density values can be converted to standard physical density units and percentage differences in density can be computed easily.

I wish to express my appreciation of the suggestions and criticisms of Dr. P. P. Werle, Dr. D. R. L. Duncan and fellow staff members of Brush Inquiry, Inc., who assisted me in the design and construction of this instrument.

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A FILING SYSTEM FOR A TEACHING COLLECTION IN ROENTGEN DIAGNOSIS

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THE following cross-index file of diseases was designed to create a teaching collection in roentgen diagnosis. The collection is used to make available illustrations of the roentgen changes in the various diseases rather than to illustrate the changes which may affect a certain region of the body. For the latter purpose the classification suggested by Hodges and Lampe[1] is preferable. On the other hand, the system which is presented here can be modified easily to suit both purposes as will be indicated later.

The method used is essentially the "library record" system. The decimal point serves to indicate major divisions. Hence 10.0 indicates the major division—The Urinary Tract. 12.0 is a major subdivision—Hydronephrosis. 12.2 represents a fairly large group—Non-calculus Hydronephrosis. 12.21, 12.22, 12.23 are further subdivisions representing the non-calculus hydronephrosis due to abnormal vessel constriction, postoperative, vesical and subvesical obstructions respectively. Assuming that the subject of "Abnormal Vessel Hydronephrosis" became more active than was anticipated, it is easily subdivided with designations 12.211, 12.212, 12.213, etc. The remainder of the classification is not disturbed in any way. Another example is afforded by the subject—Pregnancy. Since there is no obstetric service at the Mount Sinai Hospital examples of various types of pregnancy, pelvimetry, etc. are few. The entire subject is adequately designated by the code number 51.06. This part of the file is easily expanded, however, by the use of additional digits such as 51.061, 51.062, etc., or a new major subdivision 53.0 can be set up. It is essential that no films be filed in the teaching collection until the diagnosis is established with reasonable certainty by all available means.

[1] Hodges, F. J., and Lampe, I. Filing and cross-indexing roentgen-ray records. AM. J. ROENTGENOL. & RAD. THERAPY, 1939, 41, 1007–1018.

10.0 URINARY TRACT

- 10.0 Normal
  - 10.1 Normal technique
  - 10.2 Comparison of intravenous and retrograde
  - 10.3 Functional inhibition (no obvious cause)
  - 10.4 Normal physiology
  - 10.5 Extracaliceal extravasation
    - 10.51 Pyelotubular
    - 10.52 Pyelovenous
    - 10.53 Pyelolymphatic
    - 10.54 Pyeloparenchymal
- 11.0 Congenital Anomalies
  - 11.1 Position
    - 11.11 Ptosis
    - 11.12 Mobility
    - 11.13 Rotation
    - 11.14 Ectopia
      - 11.141 Pelvis
      - 11.142 Crossed
  - 11.2 Number
    - 11.21 Absence one or both
    - 11.22 Supernumerary
      - 11.221 with fusion
      - 11.222 without fusion
  - 11.3 Form
    - 11.31 Horseshoe kidney
    - 11.32 Other fusion
  - 11.4 Structure
    - 11.41 Hypoplastic
    - 11.42 Hyperplastic
    - 11.43 Polycystic
    - 11.44 Congenital cysts
  - 11.5 Renal pelvis and ureters
    - 11.51 Incomplete double ureter
    - 11.52 Complete double ureters
    - 11.53 Multiple ureters
  - 11.6 Ureteral anomalies
    - 11.61 of lumen
    - 11.62 Megalo-ureter
    - 11.63 Diverticulum
    - 11.64 Ureterocele
    - 11.65 Anomaly of implantation
  - 11.7 Vesical
    - 11.71 Absence
    - 11.72 Exstrophy

11.73 Double
11.74 Diverticula
11.75 Patent urachus
11.76 Hernia
11.8 Urethral
11.81 Epispadias and hypospadias
11.82 Congenital stricture
11.83 Diverticulum
12.0 Hydronephrosis
12.1 Calculus
12.2 Non-calculus
12.21 Pressure of aberrant vessel
12.22 Postoperative
12.23 Vesical and subvesical obstruction
12.24 Miscellaneous
12.3 Localized caliceal
12.4 Disturbed nervous mechanism
13.0 Infection
13.1 Pyelitis
13.11 Acute and subacute
13.12 Chronic
13.13 In children
13.14 In pregnancy
13.2 Pyelonephritis and pyonephrosis
13.21 Calculus
13.22 Non-calculus
13.3 Ureter
13.31 Chronic ureteritis
13.32 Stricture (post-infectional)
13.33 Ureteritis due to radium
13.34 Ureteritis cystica
13.4 Bladder
13.41 Cystitis
13.42 Perivesical
13.43 Infection due to higher bacteria
13.5 Renal suppuration
13.51 Cortical abscess
13.52 Carbuncle
13.53 Perinephric abscess
13.6 Glomerulonephritis
13.61 Nephrosis
13.7 Tuberculosis
13.71 Cortical necrosis
13.72 Pyelitis and pyonephrosis
13.73 Calcifications
13.74 Ureter
13.75 Bladder
14.0 Calculus
14.1 Parenchymal
14.2 Caliceal
14.3 Pelvic
14.4 Ureteral
14.5 Vesical and urethral
14.6 Prostatic
14.7 Differential Diagnosis
15.0 New growth
15.1 Benign Solid
15.2 Cyst
15.3 Carcinoma
15.4 Embryonal malignancy
15.5 Suprarenal tumor
16.0 Trauma
16.1 Kidney
16.2 Ureter
16.3 Bladder
17.0 Neurologic lesion of bladder
18.0 Bladder
18.1 Prostatic enlargement
18.2 Reflux
18.3 Diverticula
18.4 Neoplasm
18.5 Cystocele
18.6 Extravesical pressure
18.7 Miscellaneous
19.0 Urethra

20.0 HEART

20.0 Normal
20.1 Physiologic variations
20.2 Postural variations
20.21 Kyphoscoliosis
20.22 Kyphosis
20.3 Kymogram
20.4 Contrast visualization
21.0 Displacement
21.1 Pneumothorax
21.2 Pleural effusion
21.3 Elevated diaphragm (unilateral)
21.4 Pulmonary fibrosis
22.0 Chronic Valvular Disease
22.1 Mitral stenosis and insufficiency
22.2 Aortic stenosis
22.3 Aortic insufficiency
22.4 Combined lesions
22.5 Tricuspid disease
22.6 Pulmonic
22.7 Calcified valve
23.0 Hypertension
23.1 Essential
23.2 Chronic nephritis
24.0 Myocardial Disease
24.1 Coronary occlusion
24.11 Cardiac aneurysm
24.2 Coronary sclerosis
24.3 Myocarditis
24.4 Cardiac calcification
24.5 Trauma
24.6 New growth
25.0 Systemic Disease
25.1 Avitaminosis
25.2 Obesity
25.3 von Gierke's disease
25.4 Hyperthyroidism
25.5 Hypothyroidism
25.6 Anemia—Leukemia
25.7 Polycythemia
25.8 Arteriovenous aneurysm
25.9 Miscellaneous
26.0 Infectious Diseases
26.1 Acute rheumatic fever

26.2 Subacute bacterial endocarditis
26.3 Pneumonia
26.4 Diptheria: Scarlet fever
26.5 Miscellaneous
27.0 Congenital
27.1 Dextrocardia. Right-sided aorta
27.2 Coarctation of aorta
27.3 Patent interventricular septum
27.4 Pulmonary stenosis
27.5 Patent ductus arteriosus
27.6 Patent interauricular septum
27.7 Miscellaneous
28.0 Aorta
28.1 Normal
28.2 Widening and elongation
28.3 Syphilis
28.31 Diffuse dilatation
28.32 Aneurysm
28.321 Thoracic
28.322 Abdominal
28.323 Dissecting
28.324 Innominate artery
28.4 Atherosclerosis
28.5 Rheumatic
29.0 Pulmonary Artery
29.1 Aneurysm
29.2 Dilatation
29.21 Mitral disease
29.22 Pulmonary fibrosis
29.23 Idiopathic
29.9 Pericardium
29.91 Effusion
29.92 Pleuropericardial adhesions
29.93 Adhesive pericarditis
29.94 Calcification
29.95 Pneumopericardium
29.96 Cardiac failure

30.0–40.0 BONES AND JOINTS

30.0 Normal (except skull)
31.0 Normal development
32.0 Diseases due to prenatal influence
32.1 Congenital absence
32.2 Congenital partial absence
32.3 Accessory or supernumerary
32.4 Variations in shape
32.5 Variations in position
32.6 Fusion defects
32.7 Congenital synostosis
32.8 Chondrodysplasia
33.0 Trauma
33.1 Fracture
33.2 Dislocation
33.3 Arrest of bone following fracture
33.4 Excessive callus
33.5 Loss of substance (traumatic)
33.6 Necrosis due to roentgen rays or radium
33.7 Rarefying osteitis
33.8 Traumatic periostitis
33.9 Faulty union
33.91 Delayed union
33.92 Cross union
34.0 Infection
34.1 Bone abscess
34.2 Acute epiphysitis
34.3 Osteomyelitis
34.31 Acute
34.32 Chronic
34.33 Sclerosing
34.4 Periostitis
34.41 Acute
34.42 Chronic
34.43 Ossifying
34.5 Tuberculosis
34.51 Psoas abscess
34.6 Syphilis
34.61 Congenital
34.62 Acquired
34.7 Fungus
34.8 Boeck's sarcoid
34.9 Miscellaneous
35.0 Intoxication
35.1 Lead
35.2 Phosphorus
35.3 Radium
35.4 Bismuth
35.5 Fluorine
36.0 Circulatory Disturbances
36.1 Endarteritis
36.2 Arteriosclerosis
36.3 Thrombo-angiitis obliterans
37.0 Disturbances in Innervation
37.1 Syringomyelia
37.2 Tabes dorsalis
37.3 Raynaud's disease
37.4 Poliomyelitis
38.0 Disorder of Metabolism, Growth or Nutrition
38.1 General metabolism
38.11 Secondary hypertrophic osteoarthropathy
38.12 Disuse atrophy
38.2 Disturbance in acid-base equilibrium
38.21 Renal osteodystrophy
38.3 Disturbance in carbohydrate or lipid metabolism
38.31 Diabetes
38.32 Gaucher's disease
38.33 Schüller-Christian's disease
38.34 Miscellaneous
38.4 Vitamin deficiency
38.41 Scurvy
38.42 Rickets
38.421 Active
38.422 Healed
38.423 Deformity
38.43 Rachitis tarda
38.44 Osteomalacia
38.45 Alimentary
38.5 Endocrine disturbance
38.51 Hyperthyroidism
38.52 Hypothyroidism

38.53 Generalized osteitis fibrosa cystica
38.54 Hyperadrenalism
38.55 Disease of the testis
38.56 Acromegaly
38.57 Pituitary disturbance
38.58 Menopause
38.59 Miscellaneous
38.6 Involution Diseases
38.61 Senile osteoporosis
39.0 Disease of Unknown Cause
39.1 Osteochondritis
39.11 Vertebra
39.12 Pelvis
39.13 Carpal bones
39.14 Tarsal bones
39.15 Tibia tuberosity
39.16 Head and neck of femur
39.17 Patella
39.18 Os calcis
39.19 Osteochondritis dissecans
39.2 Epiphyseolysis
39.3 Osteitis deformans
39.31 Leontiasis ossea
39.4 Localized osteitis fibrosa
39.5 Condensing lesions
39.51 Marble bones
39.52 Condensing osteitis
39.53 Osteopoikilosis
39.6 Osteoporosis (cause unknown)
39.61 Osteopsathyrosis
39.7 Amyloid infiltration
39.8 Lymphogranulomatosis
39.9 Creeping periostitis
40.0 Joints
40.1 Anomalies
40.11 Talipes equinovarus
40.12 Hallux valgus
40.2 Infection
40.21 Specific arthritis
40.22 Arthritis of rheumatic fever
40.23 Rheumatoid arthritis
40.24 Syphilitic
40.25 Tuberculosis
40.26 Fungus
40.27 Ankylosis
40.28 Subluxation
40.3 Hypertrophic arthritis
40.31 Combined atrophic and hypertrophic
40.4 Allergic arthritis
40.5 Traumatic
40.6 Hermarthrosis
40.61 Hemophilia
40.62 Purpura
40.7 Loose bodies
40.8 Disturbance in joint meniscus
40.9 Disturbance in metabolism
40.91 Gout
40.92 Calcification in meniscus
40.95 New growth
40.96 Osteochondromatosis
40.97 Miscellaneous
40.98 Bursal calcification
41.0 Systemic Blood Disturbances
41.1 Anemia
41.11 Von Jaksch
41.12 Erythroblastic
41.13 Chronic aplastic
41.14 Hemolytic
41.2 Leukemia
41.3 Polycythemia
42.0 Tumor
42.1 Benign
42.11 Chondroma
42.12 Enchondroma
42.13 Multiple osteochondroma
42.14 Osteoma
42.15 Bone cyst
42.16 Myositis ossificans and heterotopic bone
42.17 Hemangioma
42.2 Giant cell tumor
42.3 Primary malignant
42.31 Chondrosarcoma
42.32 Osteogenic sarcoma
42.33 Ewing's tumor
42.34 Myeloma
42.35 Periosteal fibrosarcoma
42.36 Adamantinoma
42.37 Chordoma
42.4 Metastases
42.41 Osteoclastic
42.42 Osteoblastic
42.5 Lymphoblastoma
42.6 Neuroblastoma
42.7 Neurofibromatosis
42.8 Miscellaneous
43.0 Spine
43.1 Congenital
43.11 Spina bifida
43.12 Fusion of vertebral bodies
43.13 Absence of part or all of body
43.14 Klippel-Feil syndrome
43.15 Sacralization
43.16 Spondylolithesis
43.2 Disturbance of nucleus pulposus
43.21 Herniation into body
43.22 Retropulsion
43.23 Hypertrophy of ligamentum flavum
43.3 Increased lumbosacral angle
43.4 Extravertebral tumor
43.5 Spinal cord tumor
43.51 Bone destruction
43.52 Opaque myelography
43.53 Air myelography
43.6 Myelography, non-tumor
44.0 Skull
44.1 Pneumograms
44.11 Normal
44.111 Encephalogram
44.112 Ventriculogram
44.12 Congenital anomaly

- 44.13 Tumor or abscess
  - 44.131 Supra tentorial
  - 44.132 Third ventricle
  - 44.133 Posterior fossa
- 44.14 Hydrocephalus
  - 44.141 Traumatic
  - 44.142 Internal and external
  - 44.143 External
  - 44.144 Internal
  - 44.145 Epilepsy
- 44.2 Normal variations
- 44.3 Deformities sella turcica
  - 44.31 Increased pressure
  - 44.32 Extrasellar tumor
  - 44.33 Pituitary tumor
- 44.4 Localized bone destruction
- 44.5 Localized osteomatous reaction
- 44.6 Pineal shift
- 44.7 Increased convolutional markings.
- 44.8 Calcifications
  - 44.81 Brain stones
  - 44.82 Choroid
  - 44.83 Pineal
  - 44.84 Falx
  - 44.85 Tumor
- 44.9 Fracture
  - 44.91 Base
  - 44.92 Vault
  - 44.93 Depressed
- 44.94 Hydrocephalus
- 44.95 Thickening inner table
- 44.96 Osteoma
- 44.97 Foreign body
- 44.98 Salivary calculus
- 44.99 Air into orbit

45.0 Calcifications
- 45.1 Ligaments
- 45.2 Hematoma
- 45.3 Calcinosis
- 45.4 Vessels

46.0 Deformity of Undetermined Origin.
- 46.1 Skull
- 46.2 Thorax—Funnel-shape
- 46.3 Spine
  - 46.31 Cervical
  - 46.32 Dorsal
  - 46.33 Lumbosacral
- 46.4 Lower extremity
  - 46.41 Coxa vara
  - 46.42 Coxa valga
  - 46.43 Genu valgum
  - 46.44 Miscellaneous
- 46.5 Feet
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96.0 Foreign body

The system has these advantages:

(1) It is flexible. Use of the "library system" permits subheadings to be inserted or dropped without any disturbance of the main system. (2) The "break-down" or minuteness of subdivision can be as great or as small as is needed. Since the library system demands filing by numerical sequence, only as many guide cards need to be used as are required for rapid numerical localization. This depends only on the number of envelopes and not on the number of disease headings. For example, if the esophageal lesions were few, they could be filed in the numerical sequence of their code designations behind one guide card, 71.0. However, many guide cards such as 71.1, 71. 3, etc., could be used as the file becomes larger in that particular section. (3) A clerical worker with no medical vocabulary can locate any desired examples of disease in the files; the same lay person can re-insert them.

The major disadvantage lies in the fact that the files serve only as a disease cross-index. Once an examination is filed under its code designation there is no easy method, within the system, of locating it as an individual case. However, if the code designation is placed also upon the departmental file card and the teaching collection is not too large, this is not a serious drawback.

Parts of the body can be indicated in the same way that it is done in the "Standard Classified Nomenclature of Disease."[2] A dash is added after the disease code number and code numbers for the part of the body are added. We use this addition in the section on Bones and Joints. The code numbers for parts of the body are as follows:

21 Bones of Cranium, Face, and Neck (exclusive of vertebrae)
- 210 Bones of cranium and face (generally and unspecified)
- 211 Frontal bone, generally
- 212 Parietal bone
- 213 Temporal bone, generally
- 214 Occipital bone
- 215 Sphenoid bone, generally
- 216 Nasal bone; bones of nose
- 217 Malar
- 218 Superior maxilla, generally
- 219 Mandible
- 21x Hyoid bone

22 Bones of Spine and Trunk
- 220 Vertebrae
- 221 Spinous process
- 222 Lamina
- 223 Sacrum
- 224 Coccyx
- 225 Scapula
- 226 Clavicle
- 227 Sternum
- 228 Ribs
- 229 Innominate bone
- 22x Pelvis

23 Bones of Extremities
- 230 Humerus
- 231 Radius
- 232 Ulna
- 233 Carpal bones
- 234 Metacarpal bones and phalanges
- 235 Femur
- 236 Patella
- 237 Tibia
- 238 Fibula
- 239 Tarsal bones
- 29x Metatarsal bones and phalanges

24 Joints
- 240 Joints (generally and unspecified)
- 241 Vertebral joints (including atlanto-occipital)
- 242 Shoulder
- 243 Elbow joint
- 244 Wrist joint
- 255 Joints of hand and fingers
- 246 Sternoclavicular, costovertebral, chondrocostal and sacroiliac joints and symphysis pubis
- 247 Hip joint
- 248 Knee joint
- 249 Ankle joint
- 24x Joints of foot and toes

Examples are as follows: Supernumerary bones of the hands: 32.3–234; traumatic periostitis of the femur: 33.8–235.

This filing system and classification is being published because many visitors to the department felt that it suited their own pur-

[2] Published by the Commonwealth Fund, N. Y. 1936.

pose admirably. It is not likely that a classification can be devised which will meet the needs of all individuals. The present system is adapted best to a medium-sized teaching hospital where the expense of an elaborate system is not justified.

I am greatly indebted to Dr. C. B. Rabin for his help in preparing the classification of chest diseases.

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Twenty-seventh Annual Meeting: Atlantic City, N. J., 1942.

EDITORIAL

PEPTIC ULCER IN THE WAR SERVICES

THERE have accumulated over a period of years, from experimental and physiological investigations, together with numerous clinical observations, certain data which leave no doubt that there is a definite influence by the cerebral cortex over gastrointestinal movements. It is a well known fact in clinical medicine that stomach complaints develop frequently in anxiety states and other disorders "at higher levels of integration."

Recent work reported by Hesser, Langworthy and Kolb[1] on experimental data derived from stimulation and ablation procedures have further indicated a control of gastrointestinal activity by the cerebral motor and premotor cortices. Their experiments further prove or suggest that the smooth muscle of the stomach and esophagus is controlled by reflex pathways in the nervous system similar to those which control tone and contraction in the vesical muscle.

The current reports from the war hospital centers in England and the recruiting centers in Canada lend further clinical proof to the fact that the higher cortical centers play a very important part in the function of the gastrointestinal tract, and that the dyspeptics present the largest single type of disease in the Army and from several points of view "the most important medical problem of this war."

Payne and Newman[2] found that among the sick invalided back to England from France 12.5 per cent had a primary diagnosis of gastric or duodenal disease. The report of the investigation which they made on 287 cases of dyspepsia in the Army (201 from France and 86 from England) showed that this was not a new disease, as some said, or a neurosis, as others favored, but was predominantly peptic ulceration, mainly duodenal; furthermore, in nearly every case the man already had his ulcer before the war, and the cause of the immediate symptoms was due in no small part to the Army food which, though good, was too heavy for those prone to ulcers.

A recent report[3] from the National War Services Department of the Canadian Army brought out the important information that stomach and intestinal disorders stood second in the list of rejections in an examination of 10,000 men.

While it is notoriously true that all dyspeptics do not have peptic ulcer—it has been estimated that only about one in four dyspeptics actually presents, on careful study and analysis, organic lesions—it is still of great importance, not only to the wellbeing of the prospective soldier but to the future care his illness entails, that the draftees presenting symptoms of dyspepsia should have their histories gone into carefully for any previous record of a diagnosis of peptic ulcer. If such information is elicited the draftee should have the benefit of a careful study including roentgen examination before his final induction into the Army. Once in the Army he is a charge of the Army hospital doctors and when the emergency is over his health is a responsibility of the Government because "once an ulcer always an ulcer." A correct diagnosis of peptic ulcer cannot be made without careful study and examination but the majority of ulcers presenting symptoms usu-

[1] Hesser, F. H., Langworthy, O. R., and Kolb, L. C. Experimental study of gastric activity released from cortical control. *J. Neurophysiol.*, May, 1941, *4*, 274–283.

[2] Payne, R. T., and Newman, C. Quoted in *Lancet*, Jan. 11, 1941, *1*, 46–47.

[3] Editorial. Peptic ulcer—the major disability of wartime. *Canad. M. Ass. J.*, May, 1941, *44*, 508–509.

ally can be diagnosed by a careful roentgenological study, and no prospective soldier presenting symptoms clinically suggestive of peptic ulcer should be denied that study. It is true that some draftees, in order to avoid their obligations, will present themselves with complaints simulating peptic ulcer but even this type of individual, if his story rings true, should be given a careful examination before he is labelled or stigmatized as a malingerer. If the roentgenological studies definitely prove the absence of a peptic ulcer the prospective soldier should, unless there is some other vital cause for not accepting him, be inducted into a war service.

As has been aptly said, a soldier is only a civilian dressed up in a uniform and his indigestion is the same as a civilian's, not some strange disease peculiar to soldiers or sailors. Too frequently, in draftees or soldiers presenting symptoms of peptic ulcer a hasty diagnosis is made of gastric neurosis, and it is to obviate this sort of difficulty that these individuals should have a careful roentgenological examination. Therefore a definite responsibility rests upon the roentgenologist in that his examinations must be thorough and his interpretations adequate and that he be not influenced by the desires of the draftee or his immediate family in the interpretation of the roentgen findings. Only those lesions which are frankly ulcerative should be interpreted as such and the irritable gastrointestinal tract in the presence of an anxiety neurosis should not be interpreted as an organic lesion.

Peptic ulcer is a protean disease in civilian life; it is of great importance in the war services since the problem remains no longer personal, but becomes one of governmental interest. Therefore this dual responsibility must be carefully recognized by the roentgenologist.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. B. Peirce, Royal Victoria Hospital, Montreal, Canada. Annual Meeting: Cincinnati, Ohio, Sept. 23–26, 1941.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual meeting: Atlantic City, N. J., 1942.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Atlantic City, N. J. 1942.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: San Francisco, Calif., Nov. 30–Dec. 5, 1941.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Wilbur Bailey, 2007 Wilshire Blvd., Los Angeles.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES CO. MED. SOC.
Secretary, Dr. Wilbur Bailey, 2007 Wilshire Blvd., Los Angeles, Calif. Meets on second Wednesday of each month at County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. L. J. Taormina, 1093 Gates Ave., Brooklyn, N. Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. C. J. Challenger, 3117 Logan Blvd. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, J. O. Newton, 13921 Terrace Rd., East Cleveland. Meets at 6:30 P.M. at Midday Club on fourth Monday each month, October to April, inclusive.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. P. R. Weeks, 520 Republic Bldg., Denver, Colo. Meets third Friday of each month.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. J. N. Moore, 210 Professional Bldg., Ocala, Florida. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. J. C. Bell, 402 Heyburn Bldg., Louisville. Meets annually in Louisville on third Sunday afternoon in April.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society meeting. Next meeting New Orleans, April, 1942.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. J. E. Lofstrom, 1536 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. I. I. Cowan, Mt. Sinai Hospital, Milwaukee, Wis. Meets monthly on first Friday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. J. P. Medelman, 572 Lowry Medical Arts Bldg., St. Paul.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. A. O. Hampton, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. E. J. Ryan, St. Luke's Hospital, New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. H. W. Jacox, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. S. C. Davidson, 277 Alexander St. Meets on second Thursday from October to May, inclusive, 8 P.M., Rochester Academy of Medicine Building.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

St. Louis Society of Radiologists
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of October, January, March and May, at a place designated by the president.

San Francisco Radiological Society
Secretary, Dr. H. A. Hill, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months at Toland Hall, second at Lane Hall.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. L. W. Baird, Scott and White Hospital, Temple, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. C. H. Peterson, 603 Medical Arts Bldg., Roanoke, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. K. J. Holtz, American Bank Bldg., Seattle. Meets fourth Monday of each month at the College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets monthly on third Thursday from November to June inclusive, at 8:15 P.M., 32 Welbeck St., London.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine, 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1, England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. C. Singleton, Medical Arts Bldg. Toronto, 5, Ontario.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext., Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium). Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Saine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel. Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, II.52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aertze.
Permanent Secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Süd- und Westdeutsche Röntgengesellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Röntgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the spring, and one in the fall.

Societa Italiana Radiologia Medica
Secretary, M. Ponzio, University of Turin, Prof. Turin.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Măräcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime.

Society of Medical Radiology in Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen. Meets second Wednesday each month October to July in Copenhagen, at 8 o'clock in State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Roentgen Society
Meets first Wednesday of each month, at 6:30 P.M. at Zentral-Röntgen Institut des allgemeinen Krankenhauses Alserstrasse 4.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University. Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyotc, Japan. Meets bi-monthly on third Sunday.

BULLETIN OF THE INTER-SOCIETY COMMITTEE FOR RADIOLOGY

Preserving the Practice of Radiologists Called to Active Military Duty

The following communication was distributed to state councilors of the American College of Radiology and secretaries of all regional radiological societies on April 16:

The following resolution, adopted at the recent meeting of the Board of Chancellors of the American College of Radiology, is transmitted herewith for your attention:

Resolution Regarding Practice and Positions of Physicians Called to Military Service

WHEREAS, The national emergency requires the services of many physicians who have been holding hospital and other staff positions; and

WHEREAS, It is the desire of their colleagues and of all other patriotic citizens to see that the practices and positions of such men are maintained until their return insofar as such is practicable; now therefore be it

RESOLVED, That the Board of Chancellors of the American College of Radiology request regional radiological societies throughout the country to take all reasonable steps to assist colleagues called to duty and to see that the practices of those colleagues are protected as far as possible either by arranging for consultative service under conditions whereby the identity of the absent radiologist's practice is maintained or by seeing that competent assistants are appointed: and be it further

RESOLVED, That the Board of Chancellors of the American College of Radiology request the National Hospital Associations to recommend to their component state and regional units that suitable resolutions be adopted, the purport of which shall be that the maintenance of the staff or other appointments of local radiologists should be assured until their return, in good health, from military service: and be it further

RESOLVED, That the hospital physician called to duty shall, if possible, be given the opportunity to select his substitute, the appointment of whom shall be subject to the approval of the hospital authorities and staff.

In connection with hospital appointments, we call your attention to the following resolutions adopted by the Board of Trustees of the American Hospital Association on February 15, 1941:

RESOLVED: That the Board of Trustees recommend to member hospitals and other hospitals that, if consistent with local conditions, leaves of absence be granted to all employees during the period of their active military service.

While directors of hospital radiological departments are not employees of the hospital, and while the Board of Chancellors does not like to consider the appointments of staff radiologists in the same connection with employed personnel of the hospital, it is nevertheless true that the staff appointment of a radiologist presents a somewhat different problem than the positions of other physicians on the staff. With this point in mind, the Executive Committee of the Board of Chancellors conferred with the Council on Administrative Practice of the American Hospital Association during the Chancellors' meeting in February. Our Committee was assured by the Council that the resolution quoted above was meant to include the staff appointments of directors of hospital radiological departments.

The Board of Chancellors suggests that regional radiological societies consider the preparation of an agreed model contract which could be used by a member of their local society in obtaining a locum tenens for his hospital position or private office. The suggested contract should probably provide for an agreed percentage of the gross income from the practice to be retained by the locum tenens, the balance over necessary expenses to be placed in trust for the absentee. It is further suggested that the model contract contain a negative covenant by which the locum tenens agrees not to enter the practice of radiology in the community for a reasonable length of time after the absent partner returns to civil practice and the contract is terminated. The Board of Chancellors feels that, insofar as local conditions permit, each hospital radiologist who is called to service should be given the privilege of selecting his substitute or locum tenens and making the contract with the latter himself, instead of having the agreement made between the hospital superintendent and the locum tenens.

Mac F. Cahal
Executive Secretary

INDIANA ROENTGEN SOCIETY

A meeting of the Indiana Roentgen Society was held on June 1, 1941, at the Indianapolis Athletic Club. The address of the evening was given by Dr. Leo G. Rigler, of the University of Minnesota, Minneapolis, who spoke on "Cystic Disease of the Lung." The following officers were elected

for the coming year: *President:* Dr. Charles Wyeth, Terre Haute; *President-Elect:* Dr. Chester Stayton, Indianapolis; *Vice-President:* Dr. F. C. Martin, Michigan City; *Secretary-Treasurer:* Dr. H. C. Ochsner, Indianapolis.

MINNESOTA RADIOLOGICAL SOCIETY

The Thirteenth Annual Meeting of the Minnesota Radiological Society was held in St. Paul on May 27, 1941. Dr. LeRoy Sante, of St. Louis, delivered the Annual Carman Lecture before the general session of the Minnesota State Medical Association, his subject being "Roentgen Observations in Chest Injuries." The following officers were elected for the coming year: *President:* Dr. G. T. Nordin, Minneapolis; *Vice-President:* Dr. A. U. Desjardins, Rochester; *Secretary-Treasurer:* Dr. J. P. Medelman (re-elected), St. Paul.

RADIOLOGICAL SOCIETY OF NEW JERSEY

At the Annual Meeting of the Radiological Society of New Jersey held at Atlantic City on May 21, 1941, the following officers were elected for the coming year: *President:* Dr. N. J. Furst; *Vice-President:* Dr. W. James Marquis; *Secretary:* Dr. H. J. Perlberg; *Treasurer:* Dr. H. A. Vogel; *Counsellor:* Dr. Charles Oderr.

AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians announces that its Twenty-sixth Annual Session will be held in St. Paul, Minnesota, on April 20–24, 1942. Dr. Roger I. Lee, of Boston, is President of the College and will be in charge of the program of General Sessions and Lectures. Dr. John A. Lepak, of St. Paul, has been appointed General Chairman, and will be in charge of the Program of Hospital Clinics and Round Table Discussions, as well as local arrangements, entertainment, etc. Mr. Edward R. Loveland, Executive Secretary of the College, 4200 Pine St., Philadelphia, will have charge of the general management of the session and the technical exhibits.

AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology will hold its fall examination in Cincinnati, Ohio, on September 27, 28 and 29, 1941. There will be another examination in Atlantic City in June, 1942. Those wishing to be examined in Cincinnati should file their applications immediately.

B. R. Kirklin, Secretary
The Mayo Clinic
Rochester, Minnesota

AMERICAN ROENTGEN RAY SOCIETY

It was intended that the schedule of the Instruction Courses to be given at the coming meeting of the American Roentgen Ray Society in Cincinnati, September 23–26, 1941, be published in this issue of the JOURNAL. Due to an unforeseen delay it is not possible to do this. However, both the schedule of the Instruction Courses and the Preliminary Program of the meeting will appear in the August issue.

MIDSUMMER RADIOLOGICAL CONFERENCE

The following is the program of the Seventh Midsummer Radiological Conference in the Rocky Mountains to be held in Denver, Colorado, July 31, August 1 and 2, 1941. All meetings will be held in the Hotel Shirley-Savoy. There is no registration fee.

Guest Speakers

(As they appear on the program)

John D. Camp, M.D., Associate Professor of Radiology, Mayo Foundation, Rochester, Minnesota

John T. Murphy, M.D., Director, Roentgen Department, St. Vincent's Hospital, Toledo, Ohio

Ross Golden, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia University, New York City

David S. Beilen, M.D., Vice President, Radiological Society of North America, Chicago, Illinois

Leon J. Menville, M.D., President-elect, Radiological Society of North America, New Orleans, Louisiana

U. V. Portmann, M.D., Director, Department of Therapeutic Radiology, Cleveland Clinic Foundation, Cleveland, Ohio

Wendell G. Scott, M.D., Assistant Professor of Radiology, Washington University, St. Louis, Missouri

Thursday Afternoon, July 31

12:00 to 2:00 P.M. Registration

2:00 P.M. Conference called to order. Elizabeth H. Newcomer, M.D., Presiding, President, Denver Radiological Club

Address of Welcome. William H. Halley, M.D., President, Colorado State Medical Society, Denver

2:15 P.M. "Roentgenologic Observations Concerning Osteoporosis and Its Relation to Systemic Diseases," John D. Camp, M.D., Rochester, Minnesota

3:15 P.M. "Vascular Tumors of Bone," Atha Thomas, M.D., Denver

4:00 P.M. "Bone Tumors," John T. Murphy, M.D., Toledo, Ohio

5:00 P.M. General Discussion, Kenneth D. A. Allen, M.D., Denver, Lloyd R. Allen, M.D., Colorado Springs, Ernst A. Schmidt, M.D., Denver

Thursday Evening, July 31

8:00 P.M. Joint meeting with the Medical Society of the City and County of Denver, Leonard G. Crosby, M.D., Presiding, President of the Medical Society of the City and County of Denver

Symposium on Gastro-intestinal Tract

Esophagus and Stomach, John D. Camp, M.D.

Small Intestine, Ross Golden, M.D.

Colon, David S. Beilen, M.D.

Symposium on Irradiation of Malignancy of Breast

Preoperative Irradiation, Leon J. Menville, M.D.

Postoperative Irradiation, U. V. Portmann, M.D.

Discussion

Wendell G. Scott, M.D.

John T. Murphy, M.D.

Dutch lunch following program

Friday Morning, August 1

9:00 to 10:00 A.M. Organization Meeting

10:00 A.M. "Body Section Radiography as Aid in a Diagnosis of Thoracic Aneurysm or Mediastinal Tumors," Wendell G. Scott, M.D., St. Louis

11:00 A.M. Discussion, Cassie Bell Rose, M.D., Boulder, George Unfug, M.D., Pueblo

11:15 A.M. "Roentgenology of the Small Intestine," Ross Golden, M.D., New York City

12:15 P.M. Adjournment

12:30 P.M. to 2:15 P.M. Round Table Luncheon, Discussion on Diagnostic Problems, Leonard G. Crosby, M.D., Denver, Presiding

Friday Afternoon, August 1

2:30 P.M. "Roentgenology of the Small Intestine," Ross Golden, M.D., New York City

3:30 P.M. "Roentgen Therapy in Non-Malignant Conditions," D. S. Beilen, M.D., Chicago

4:00 P.M. "Irradiation for Diseases of the Thyroid Gland," U. V. Portmann, M. D., Cleveland

5:00 P.M. General Discussion, Edward J. Meister, M.D., Denver, Harmon Brandenburg, M.D., Denver, Frank B. Stephenson, M.D., Denver

Friday Evening, August 1

7:00 P.M. Social Hour

7:30 P.M. Informal Banquet, Elizabeth H. Newcomer, M.D., Presiding, President, Denver Radiological Club

8:30 P.M. Introduction of Guests

9:00 P.M. Dancing

Saturday Morning, August 2

9:00 A.M. "Carcinoma of the Skin," John T. Murphy, M.D., Toledo

10:00 A.M. "Factors Which Influence the Curability of Malignant Tumors," U. V. Portmann, M.D., Cleveland

11:00 A.M. "Irradiation Therapy in Endocrine Dysfunction," Leon J. Menville, M.D., New Orleans

12:00 P.M. General Discussion, W. W. Wassan, M.D., Denver, N. B. Newcomer, M.D., Denver

12:15 P.M. Adjournment

12:30 P.M. to 2:30 P.M. Round Table Luncheon, Discussion of Therapeutic Radiology, Paul R. Weeks, M.D., Denver, Presiding

BOOK REVIEWS

Books sent for reviews are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers as space permits.

Clinical Roentgenology of the Alimentary Tract. By Jacob Buckstein, M. D., Visiting Roentgenologist (Alimentary Tract Division), Bellevue Hospital, New York City; Consultant in Gastro-Enterology, Central Islip Hospital. Cloth. Price, $10.00. Pp. 652, with 525 illustrations. Philadelphia & London: W. B. Saunders Co., 1940.

In this volume, the author has presented in well-arranged order and in considerable detail facts regarding the roentgen diagnosis of diseases of the alimentary tract. This is in the main a reflection of his wide personal experience but he also draws freely from original sources for the development of the historical background which begins each chapter. The book is illustrated with cuts of the author's cases which are well selected and excellently reproduced. His discussion of diseases of the small intestine is good but it would be enhanced by more specific reference to the details of the use of the Miller-Abbott tube or its equivalent. Excellent chapters on the gallbladder and pancreas are a valuable part of the work. The volume is well worth the careful study of any roentgenologist or gastroenterologist, and would be a valuable addition to his library.

H. Dabney Kerr

The 1940 Year Book of Radiology. *Diagnosis:* Edited by Charles A. Waters, M.D., Associate in Roentgenology, Johns Hopkins University; Assistant Visiting Roentgenologist, Johns Hopkins Hospital; Associate Editor, Whitmer B. Firor, M.D., Assistant in Roentgenology, Johns Hopkins University; Assistant in Roentgenology, Johns Hopkins Hospital. *Therapeutics:* Edited by Ira I. Kaplan, B.Sc., M.D., Director, Radiation Therapy Department, Bellevue Hospital, New York City; Associate Radiologist, Lenox Hill Hospital, New York City; Clinical Professor of Surgery, New York University Medical College. Cloth. Price, $5.00. Pp. 496, with 497 illustrations. Chicago: The Year Book Publishers, Inc., 1940.

To those well established in the practice of radiology an annual review of the Year Book is unnecessary for they are familiar with its form and expect that it will contain a synopsis of all important contributions to radiology for the current year.

For those embarking on a career or just becoming interested in the subject there is no better volume than that edited by Waters, Kaplan, and Firor.

The 1941 edition is, no doubt, well under way and has a high standard to maintain if it approaches the excellence of the 1940 edition. Special features of this "Anniversary" number were: a history of the Year Book and special articles by both Waters and Kaplan, and photographs of the three editors which are of interest to those who do not know them personally, and are appreciated by their friends.

Waters, Firor and Kaplan have ably carried a burden which becomes increasingly difficult every year as the volume of radiological literature increases. Even in this year of turbulent European affairs a surprising number of European articles are abstracted. Attention is given to papers dealing with subjects of military interest such as war wounds and foreign bodies, and Kaplan calls attention to the numerous uses for radiation therapy in war time such as post-surgical parotitis, furuncles and carbuncles, gas bacillus infection, and very importantly in prophylaxis in abdominal surgery and extensive war wounds. Ununited fractures and extensive scars and contractures are also among the surgical needs for irradiation in military medicine.

Other features of special interest are arthrography in ruptures of tendons of the shoulder joint, post-traumatic atrophy of painful joints, foreign bodies associated with arthritis and fate of cranial defects due to fractures and surgery.

The respiratory system is represented by numerous items of interest. Extrapleural pneumolysis is intriguing in its possibilities of replacing extensive thoracoplasty. Paraffin plombage has a similar purpose. Tracheobronchial tuberculosis, bronchomycosis and nonspecific bronchial infections are discussed. It is impossible to catalogue all the interesting articles in the divisions dealing with the glandular, gastro-

intestinal, vascular and genitourinary systems but the reader is well repaid for a few minutes spent in glancing over these sections.

Kaplan's special article on amenorrhea and sterility is backed by years of experience and there is food for thought in his statement that, "irradiation, when properly given, is harmful neither to the mother nor to the offspring and has proved a valuable therapeutic procedure for treatment of amenorrhoea and the relief of sterility." His introduction to the therapeutic section well sums up the present ideas of therapy and their relation to the current and possible future developments.

Those who have not yet secured this book will render themselves a service by doing so at once and be prepared for the new feast of radiological knowledge which will be forthcoming in the 1941 edition.

E. Walter Hall

The Calcified Pineal Gland. By Dr. W. Bergmann. Paper. Pp. 89, with 11 illustrations. Assen: Van Gorcum & Comp. N. V., 1940.

This is a short monograph on the pineal gland. In the brief introduction the author states, "In order to be able to carry out my intention of establishing a connection between the calcification and the pathology of the pineal gland, I had also to study the histology in great detail," which he does in four pages.

Chapters I, II, and III are devoted to the embryology, histology, and anatomy of the pineal gland.

Chapter IV discusses the various methods of localizing the calcified pineal body. This chapter is of interest to the roentgenologist for the author reviews the literature on the displacement of the pineal body as reported by Schüller, Naffziger, Vastine and Kinney, Dyke, and Fray.

Chapter V is devoted to "confusion" of the pineal with other structures. Among such calcified structures which might be confused with the pineal, he names the choroid plexus and the falx, and he sets forth the well-known criteria for differentiating the latter from the pineal.

The sixth section of the monograph deals with the percentage of calcified pineals in the various decades, and the author confirms the fact that calcification in the pineal increases in frequency in direct proportion to the patient's age.

Chapter VII is devoted to a discussion of the calcification and weight of the epiphysis. During the first twenty years, the pineal weight-increase is almost entirely due to development of parenchyma, while between the twentieth and fortieth years the increase in weight is largely due to the deposition of calcium, while the decrease in weight of the pineal after the fiftieth year, he believes, is due to a reduction of the parenchyma.

The eighth section treats of the form of calcification in the pineal gland. He remarks that Schüller (1909) was the first to interpret a shadow in the roentgenogram as the pinealis and proved it by dissection. The author emphasizes that the propineal is often calcified before the actual pineal. The calcification in the propineal is hook-shaped and lies anterior to the actual calcium in the pineal body. Furthermore, he points out that the calcification is generally in the apex and only in the glandular portion, not in the glial portion of the pineal body. He believes that the extent to which the gland is calcified depends upon the proportion of the parenchyma to the other portions of the gland. He suggests that calcification of the parenchyma of the gland or of the whole organ is noticeable in pathological disturbances of the vessels, whereas a calcification of the propineal body has no pathological significance.

In Chapter IX, the author discusses the physiology of the pineal gland and he is an enthusiastic supporter of the view that the pineal body is an organ with internal secretion. He then reviews the experimental work that has been performed on the pineal.

The tenth section is devoted to the pathology of the pineal body. He further states that his own cases of "strongly" calcified pineals, which he attributed to hypofunction, showed certain characteristic anomalies such as: (1) Vascular disturbances of perspiration, congestion, paresthesia of the hands and feet associated with cutis marmorata, arteriosclerosis praecox, hypertension, and Burger's disease. (2) Psychic disturbances—by that term he means the patients are always nervous. (3) Disturbances of sleep. (4) Sexual disturbances.

Chapter XI describes the significance of the presence of a calcified pineal in the diagnosis and therapy of disturbances of the pineal function. He states that hypofunction of the pinealis in youth produces hypertrophic growth, hypergenitalismus, and hypertrichosis.

Chapter XII tells of the percentage of calcification in the pineal gland in relation to diseases, and the latter he divides as follows:

(1) Neurological anomalies
Intracranial tumors
Commotiones
Skull traumata and fractures
Epilepsy
N.a. sensu strictiori
(2) Psychic anomalies
(3) Disturbances of the vascular regulation
(4) Other physical anomalies.

Under neurological anomalies he first considers intracranial tumors of which he has 175 cases, so diagnosed clinically. In 25 per cent of the cases the diagnoses were confirmed by dissection. In regard to tumors, the incidence of calcification in the pineal parallels the normal curve up to the thirtieth year, but after that the percentage of calcification is far below the normal standards. Towards the seventieth year the two curves tend to approach each other again. The incidence of calcification of pineals in commotiones and skull fractures when compared with the standard graph shows that until the fortieth year they run practically parallel. From the fortieth to the fiftieth year the former runs above the standard graph, and at about the sixtieth year the two graphs meet again. There are 100 cases in this group. In the epileptic group there are 128 cases, and the incidence of calcification in the pineal was parallel until the thirtieth year and after that it was higher in the epilepsy graph.

In the psychic diseases, and the author subdivides these which it is not possible to do here, the incidence of calcification in the pineal is strikingly higher than in normal individuals.

In the group with disturbances of vascular regulation, there are 57 cases and the incidence of calcification in the pineal is much higher then in the standard normal series in the various age groups.

Chapter XIII is a discussion of therapeutic results of pineal medication, and in Chapter XIV, fifteen short case reports are given.

Chapter XV is a summary of the monograph.

This monograph should be of interest to radiologists, neurologists, psychiatrists, and endocrinologists. It is extremely concise and the author has opened the door to further investigation of the significance of calcium in the pineal body.

C. G. Dyke

Les Bases experimentales de la Radiothérapie totale et subtotale. Par Yu-Hans T'ang, Docteur en Médecine, Docteur spécial en Médecine, Ex-interne à l'Institut du Cancer de Louvain. Paper. Pp. 93, with several illustrations. Brussels: Imprimerie Médicale et Scientifique (S.A.), 1939.

This research was carried out in the Cancer Institute of the University of Louvain, J. Maisin, Director, on thirty rabbits which the author treated with various doses and types of radiation. He found that doses of 100 to 150 r administered to the whole body given at a rate of 10 to 25 r a day are not dangerous. Doses over 200 r produce leukopenia; the dangerous dose is 500 r given at one time and 5,000 to 6,000 r at the rate of 10 to 25 r a day of total radiation. Contraindications to continuance of treatment are leukopenia with anemia. Vitamin C decreases the radiosensitiveness of an organ; diformaldehyde peroxid increases it; vitamin B_1, B_2 and B_6 have no effect. The author next cites some clinical applications of the method, making no reference to the works of Teschendorf, Heublein or Scott, in a short, largely French bibliography.

E. T. Leddy

Biological Symposia. Volume I. Edited by Jaques Cattell, Editor of The American Naturalist and American Men of Science. With a Foreword by Albert F. Blakeslee, Director of the Department of Genetics, Station for Experimental Evolution of the Carnegie Institution of Washington and President of The American Association for the Advancement of Science. Cloth. Pp. 238, with illustrations. Lancaster, Pa.: The Jaques Cattell Press, 1940.

Obvious to all thoughtful students of biology is the pressing need, nowadays, for authoritative summarizations of the advances and prevailing ideas in the various fields. *Biological Symposia* makes an auspicious start to satisfy this need. The volume contains three symposia that were presented in December, 1938, at the Richmond, Virginia, meeting of the American Association for the Advancement of Science. The first deals with the cell theory, the second with mating types and their interactions in the ciliate infusoria, and the third with chromosome structure. The expositions presented by seventeen outstanding investigators make stimulating reading and give clear views of the early developments and the present status of the problems under discussion.

Barnett Cohen

DEPARTMENT OF TECHNIQUE

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A TECHNIQUE FOR THE MORE PRECISE LOCALIZATION OF PULMONARY ABSCESSES*

By COLEMAN B. RABIN, M.D.
NEW YORK, NEW YORK

THE recent report by Neuhof and Touroff[1] of the cure of putrid abscess of the lung by a one-stage drainage operation during the acute phase will undoubtedly lead to the institution of this method of treatment by many surgeons. It must be emphasized that if the remarkable results obtained by these workers are to be duplicated, or improved upon, not only must a meticulous technique be observed, but the utmost accuracy in the location of the abscess must be achieved. It is necessary to determine roentgenologically where the abscess is most superficial, for in this small area are situated the pleural adhesions through which the drainage of the lesion is to be accomplished. A small margin of error may lead to an opening of the pleura, thus transforming a relatively simple operation into a complicated procedure with an attendant increased mortality.

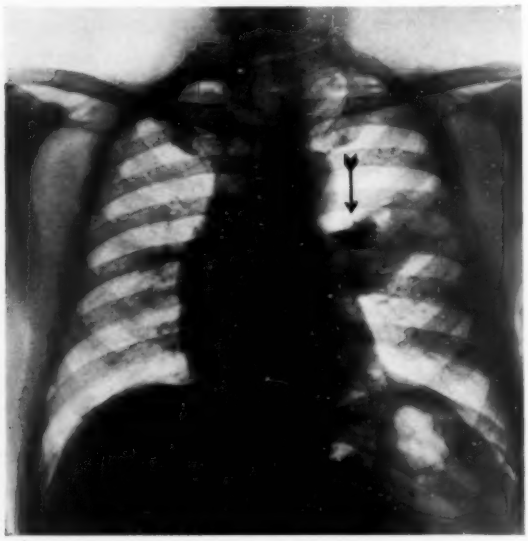

Fig. 1. Abscess in anterior segment of left upper lobe at level of fourth rib anteriorly. The spot of iodized oil and methylene blue is situated in the third intercostal space over the mesial border of the abscess. The lesion was drained after the resection of a segment of the fourth rib, just lateral to the landmark. Were it not for the landmark, the wrong rib might have been resected because the obesity of the patient precluded proper rib counting and the anterior prominence of the chest was caused by the third rib cartilage instead of the second as is ordinarily the case.

By means of roentgenography in the dorsoventral, lateral and oblique positions, together with roentgenoscopy, an accurate idea of the location of the lesion may be obtained in almost all cases of acute abscess of the lung.[2] It is essential, however, that this knowledge of the location of the abscess be utilized by the surgeon in a precise manner when he attempts to approach the lesion. In our experience this has been at times a difficult problem. Although the rib level of the abscess has been determined roentgenologically, there is often no landmark visible in the limited operative wound to aid the surgeon in selecting the proper segment of that rib for resection. Moreover, the frequency of rib variations and the difficulty of palpating the ribs through the overlying tissues often make rib identification difficult and uncertain. These factors prevent the full utilization, by the surgeon, of the knowledge gained from the roentgen studies of the location of the abscess, so

* From the Group for Thoracic Diseases and the Department of Radiology of the Mount Sinai Hospital, New York.

that he often finds himself uncertain of the location of the lesion in relation to his incision.

In order to obviate these difficulties, the following method was devised: There is introduced, into the intercostal muscle over the region of the abscess, a marker that

Table I

Position of Abscess	Roentgen Position Recommended
Mesial half of chest	Dorsoventral or ventrodorsal
Right anterolateral Left posterolateral	Right oblique
Left anterolateral Right posterolateral	Left oblique
Mid lateral (between the anterior and posterior bends of the ribs)	Lateral

Showing the proper roentgen views for "landmark" localization of variously situated abscesses. In these views there is the least distortion of the distance between the landmark and the abscess.

serves as a landmark, visible both on the roentgen film and in the operative wound before rib resection. This is accomplished by injecting a mixture of 0.2 cc. each of iodized oil and 0.5 per cent methylene blue into the intercostal muscle as near as possible to the site of the abscess, as localized previously by appropriate roentgen methods. The injected mixture appears on the roentgen film as a radiopaque landmark, usually showing striations produced by the fibers of the intercostal muscle (Fig. 1). The relationship of the center of the abscess to this landmark is determined from the roentgen film, made in a position in which there is the least distortion of the distance between the landmark and the abscess (Table 1). Once this relationship is known, it remains a comparatively simple matter for the surgeon to determine the site for his rib resection. The landmark that has been established is recognizable, after the superficial incision has been made, by the blue discoloration of the intercostal muscle. The selection of the portion of rib to be resected is judged from the relationship of the landmark to the abscess as determined from the roentgen film.

This method of localization has been employed in 21 cases of abscess of the lung, and has been found to be a most useful and reliable guide in locating the lesions at operation. In fact, in the case of abscesses situated in the outer half of the lung, I regard the method as indispensable, and even in abscesses situated in the mesial half of the chest the use of the method has obviated the possibility of error in the choice of the site for drainage.

SUMMARY

A method is described for the establishment of a landmark, visible on roentgen films and at operation, to aid in the location of pulmonary abscesses. This consists of the injection of a small quantity of a mixture of methylene blue and iodized oil into the intercostal muscle over the region of the abscess. The relationship of the injected spot to the abscess is determined by roentgen examination in the appropriate position. This relationship is utilized by the surgeon to determine the site for drainage of the abscess after locating the blue spot in the intercostal muscle at operation.

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2. Rabin, C. B. Localization of abscess of the lung, with special reference to the oblique view. To be published.

Since this article was written the method has been used, on the Orthopoedic and Neurological Services of the Mount Sinai Hospital, for the identification of the vertebrae at laminectomy by the injection of the mixture between the spinous processes and the study of lateral views of the spine before the operation.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

BRODY, BERNARD S. The management of acute cranio-cerebral injuries. *J. Am. M. Ass.*, Sept. 21, 1940, *115*, 977–983.

The great majority of patients with head injuries will recover with ordinary bed rest supplemented in some instances by lumbar puncture or hypertonic solutions. Those patients with an uncomplicated concussion should be kept in bed for approximately ten to fourteen days. Roentgenograms of the skull, when needed for medicolegal reasons or otherwise, can be taken when the patient's condition warrants the effort. A diagnostic spinal puncture should be done at some time within the first week for patients whose spinal fluid is bloody or whose period of unconsciousness is prolonged, the diagnosis of simple concussion alone is no longer tenable and the period of bed rest must be proportionately longer.

Responsibility to the patient with a cranial injury resolves itself into three categories: the prevention of infection, the treatment of increased intracranial pressure and the management of depressed fractures.

Prompt and efficient attention should be paid to lacerations of the scalp. The patient who has definite evidence of a fracture through the mastoid or paranasal sinuses in the form of cerebrospinal otorrhea or rhinorrhea should be started promptly on sulfapyridine as a preventive measure, because the pneumococcus is the most common invading organism. In cases with persistent bleeding from the nose or ear in which it is difficult to determine whether an admixture of spinal fluid is present or not, the laminagraph has been of considerable help. From the standpoint of prevention of infection, the practice of elevating the foot of the bed about 12 inches has been of material help in decreasing the incidence of aspiration pneumonia in patients who are in deep coma. A compound depressed fracture calls for prompt débridement of devitalized tissues.

The treatment of increased intracranial pressure revolves about the diagnosis. Those patients who present the classic symptoms of extradural or subdural bleeding will die almost invariably unless the clot is removed. The second lapse into unconsciousness is due to cerebral compression from the clot. In the group of patients who do not present this classic picture and yet harbor a definite clot, the lucid interval is missing for the reason that there is, in addition to the clot, extensive underlying brain damage. These cases constitute the group in which multiple trephine holes are indicated. In this way a clot may be located and a life saved if the underlying cerebral damage is not too extensive. Those patients who are severely injured and persist in showing an elevated spinal fluid pressure over 200 mm. of water, in the event that no clot is found should have a generous decompression if the brain appears to be under serious pressure. A thorough neurologic examination of the patient on admission and at frequent intervals thereafter is strongly emphasized.

Every scalp laceration should be carefully examined under sterile precautions before being sutured, to establish the presence or absence of an underlying fracture. Further information concerning the nature of the fracture, if it appears grossly to be depressed, should be obtained by roentgen examination before the wound is closed. A compound depressed fracture must be treated promptly in the manner discussed. Only occasionally will a patient be in such a degree of shock from gross tearing of the brain as to be unfit for any immediate major surgery. Late epileptic manifestations are due to the formation of a cerebral cicatrix rather than to any irritative effects of a depressed fragment. Early elevation of the depression apparently does not prevent the development of late sequelae. The primary objective in the operative treatment is the débridement of devitalized tissues and the repair of the cranial deformity.

Occasionally patients are so severely injured that death occurs within a few hours. Some of them, in spite of the extremely ominous picture which they present, survive the immediate critical period and gradually return to consciousness after days or weeks. The prolonged period of unconsciousness is due to widespread

cerebral lesions in the nature of multiple lacerations and contusions, or petechial hemorrhages or extensive hemorrhage into the brain stem from the vein of Galen with associated edema and cell damage. It is this group in which the method of repeated lumbar punctures and conservative use of hypertonic solutions has its best clinical application.

According to Glaser and Blaine, roentgenograms of the skull taken from six months after injury in children to nine months after injury in adults are as valuable as those taken immediately after injury in determining the presence or absence of fracture. The indications for immediate roentgenograms are few. Compound fractures and fractures through the sinuses are examples of cases where roentgenograms should be made at once.

It appears wiser to refrain, if possible, from the use of a lumbar puncture during the first twenty-four hour period. This is based on the assumption that intracranial hemorrhage might be accelerated by the rapid removal of cerebrospinal fluid. The spinal fluid pressure may at times be misleading tending gradually to return to normal, whereas the patient is actually becoming more drowsy, probably due to either an incisural or a foraminal block.

Extreme restlessness may precede or accompany a state of progressive stupor due to increasing intracranial pressure, and the tendency to complicate the picture with sedation may make the differentiation difficult. Restlessness can be controlled in part by repeated lumbar punctures. Morphine in particular is contraindicated because of its depressing effect on the vital centers. Headache can usually be controlled by acetylsalicylic acid. Emphasis is laid on the fact that the classic signs of increasing intracranial pressure, and especially a notable rise in blood pressure with a concomitant increase in pulse pressure, may be a relatively late development in the clinical picture. If one waits for a striking rise in blood pressure the patient may not survive even if a blood clot is successfully removed. A tendency to extreme restlessness and increasing stupor is an earlier and more reliable indicator of increasing intracranial pressure than the vital signs.—*S. G. Henderson.*

BUCY, PAUL C., and HAVERFIELD, W. TRACY. Cranial and intracranial complications of acute frontal sinusitis. *J. Am. M. Ass.*, Sept. 21, 1940, *115*, 983–991.

The most common complications of acute frontal sinusitis, particularly that which follows swimming, are abscess of the frontal lobe and osteomyelitis of the frontal bone. The clinical course in which an intracerebral abscess develops from the ear or from the paranasal sinuses is typical. The intracranial infection is heralded by the general manifestations of increasing infection. The temperature rises, there is an increasing leukocytosis, the patient is "toxic." He may have a chill and almost invariably there is rigidity of the neck. This sudden exacerbation may last only from one to three days. After a variable period of days or weeks, signs of cerebral or cerebellar involvement appear. Now the usual evidences of infection, fever, tachycardia and leukocytosis are commonly minimal or absent. Shortly after the appearance of this second stage, evidences of increasing intracranial tension, headache, vomiting and choking of the optic discs, develop. Blindness, stupor, coma and death may follow.

There are several potential avenues by which the infectious organisms may invade the frontal bone and the intracranial cavity. The pathway most commonly indicted is the diploic veins. Spreading upward from the region of the frontal sinus into the frontal bone and to emissary veins which penetrate to the superior longitudinal sinus or its immediate tributaries are two diploic veins, the frontal and the anterior temporal. Osteomyelitis develops only when thrombosis occurs and in some instances infection may pass through these veins into the superior longitudinal sinus and thence, retrograde, into the ipsilateral or contralateral cerebral hemisphere without the venous wall becoming infected.

The most important problems in acute frontal sinusitis are (1) prevention of the sinusitis and (2) treatment of the sinusitis, once it develops, adequate to prevent the appearance of complications. The worst cases of frontal sinus infection and osteomyelitis have followed swimming. Prevention of acute frontal sinusitis is best brought about by the teaching of proper breathing, the use of suitable devices, such as nose clips and ear plugs, while swimming, and the avoidance of chilling.

In fulminating cases of frontal sinusitis associated with pain, tenderness, local swelling and fever, particularly those cases which follow swimming, adequate external drainage of the infected sinus becomes imperative on making the diagnosis. Endonasal drainage is wholly inadequate for this purpose. Drainage must be

prompt and must be obtained before any extension of the infection beyond the sinus has occurred.

Once progressive osteomyelitis has set in, the only chance of saving the patient lies in the immediate and entire removal of the diseased bone. Osteomyelitis does not give roentgenographic evidence of its presence for at least ten to fourteen days after the onset. Fortunately there is one constant and pathognomonic sign of osteomyelitis of the frontal bone, an edema of the forehead extending upward from the supra-orbital ridge. A brain abscess should be approached through uninfected tissue if possible. Usually the defect in the skull caused by removal of infected bone heals by spontaneous regeneration of bone.—*S. G. Henderson.*

Maresch, Marion M., and Washburn, Alfred H. Paranasal sinuses from birth to late adolescence. II. Clinical and roentgenographic evidence of infection. *Am. J. Dis. Child.*, Oct., 1940, *60*, 841–861.

The authors' material for this paper consisted of data collected on average, healthy children who had roentgenograms taken at regular intervals, without regard to symptoms or signs of disease of the sinuses. They have found in the roentgenograms all variations usually attributable to infection without being able to prove any consistent or definite relation between the pattern of development of the sinuses and the incidence of infections.

The cross sectional analysis of the 3,501 roentgenograms of the sinuses of the 100 children who make up the study demonstrated that a relatively constant percentage taken between one year and six years of age showed "pathologic" antrums, that is between 32 and 35.5 per cent. After the age of six years the incidence of "pathologic" antrums dropped steadily until it showed about 15 per cent at the age of twelve.

In 72 children who were followed from the first year of life to the present time, 30.7 per cent showed "pathologic" antrums. The authors could find no consistent causal relationship between the occurrence of asymmetric antrums and the roentgenographic evidence of infection of the sinuses. Although no correlation between the size of the antrums or of the developing frontal sinuses and the incidence of "pathologic" antrums could be shown, the authors do not imply that infection never influences the growth of the sinuses. They do wish to imply that variations in size of the sinuses occur frequently, without any demonstrable relation to the amount or frequency of infections as seen in the roentgenogram and without following any definite pattern.

Tonsillectomy had no demonstrable effect on the roentgenographic appearance of the sinuses. The children with definitely allergic manifestations (26 per cent of 100 children) showed a higher percentage of "pathologic" sinuses up to six years of age than did the nonallergic group, but after that age there was no essential difference between the data of these two groups.

The appearance of the series of films of the chest of each child is the basis for the authors' belief that the relation of infection of the sinuses to possible disease of the chest has been erroneously overemphasized in the literature. The range of "normal" for the roentgenographic appearance of the chest is probably much wider than one has been led to expect. Differences in the roentgenograms of the chest of different children demonstrate that the appearance of a child's chest is established during the first few months of life and bears no consistent relation to the amount of infection of the sinuses. The "normal" or optimum appearance for one child is not necessarily the same as that for another child. From the results of this study, they conclude that it seems probable that the size and pattern of growth of a child's sinuses and the appearance of the hilar shadows and pulmonary markings in the roentgenograms of his chest are just as much inherent individual characteristics as are the shape of his nose or the color of his eyes.—*R. S. Bromer.*

Warkany, Josef, and Weaver, Thomas S. Heredofamilial deviations. II. Enlarged parietal foramens combined with obesity, hypogenitalism, microphthalmos and mental retardation. *Am. J. Dis. Child.*, Nov., 1940, *60*, 1147–1154.

A case is reported, of a negro boy, eleven and one-half years of age, who had enlarged parietal foramina combined with obesity, disturbance of carbohydrate metabolism, hypogenitalism, microphthalmos, and mental retardation. The authors consider these abnormalities as coordinated developmental defects. They point out that some of these defects are present in the Laurence-Moon-Biedl syndrome, while others have not been seen in this combination. Their review of the literature revealed that en-

larged parietal foramina occur frequently in children who have other developmental abnormalities.

Defects in the bones of the skull are seen in association with meningo-encephalocele, syphilis, xanthomatosis, multiple myeloma, multiple malignant metastasis, renal and celiac rickets, cleidocranial dysostosis, and after trauma or opening for ventriculographic examination. However, if one bears in mind the possible occurrence of enlarged parietal foramina, usually symmetrically placed on each side of the posterior third of the sagittal suture and irregularly shaped, with well defined margins, the diagnosis is relatively easily made. Diagnosis of the condition in a newborn infant is sometimes difficult, owing to the fact that a single large defect may extend across the sagittal suture and into both parietal bones.—*R. S. Bromer.*

Bronstein, I. P., and Fabricant, Noah D. Pituitary dwarfism with atrophic rhinitis. *Am. J. Dis. Child.*, Nov., 1940, *60*, 1140–1146.

Children exhibiting sexual infantilism and defective growth (Lorain-Léri dwarfs) may be normal at birth and develop adequately until some time in childhood when growth either ceases or becomes retarded. The arrest involves no special organs and produces a diminutive child whose proportions are normal. The genital retardation is probably not in excess of that of the physical development, but the secondary sexual characteristics are markedly delayed or absent. The mentality may be normal, although the behavior is often immature. This type of dwarf, as a general rule, is osteologically normal, although the skeletal framework is small. However, chondro-epiphysial changes, poorly outlined centers of ossification with decreased density of the bone have been described. The centers of ossification usually appear at the normal time, whereas delay in epiphysial closure is found consistently.

The diagnosis of pituitary dwarfism is certain only in those cases in which (1) the hypophysis either has been removed or is known to have been destroyed, necropsy verifying the diagnosis; (2) the roentgenogram demonstrates the presence of a tumor of the pituitary gland, or (3) definite and continuous response to administration of growth hormone occurs.

In the case reported in this paper, a girl, first seen at the age of twelve years, a calcified lesion in the sella turcica was found on roentgenographic examination. She had also a marked atrophic rhinitis. Estrogens, which have been employed by others in treatment of atrophic rhinitis, were not used in this patient because of the authors' fear of possible premature epiphysial closure. The irregular administration of growth-promoting substances over a period of approximately two and one-half years was presumably responsible for a gain in weight and an increase in height but had no apparent effect on the atrophic rhinitis.—*R. S. Bromer.*

NECK AND CHEST

Camplani, Mario. Fistola esofago-cutanea e diverticolo di Zenker. (Esophago-cutaneous fistula and Zenker's diverticulum.) *Radiol. med.*, July, 1940, *27*, 581–583.

A woman of twenty-five came for examination for a lesion of the skin on the left side of the neck two fingerbreadths above the clavicle. There was a reddish, elevated button of skin, ulcerated at the center with a scanty discharge of serous fluid. There were no enlarged glands. Evidently it was a fistula. The patient said that for a long time she had had some difficulty in swallowing and that a few years ago she had swallowed a bone. After some time the fistula had developed.

Roentgen examination showed a small sac just below the mouth of the esophagus, just beneath the laryngeal cartilage at the level of the seventh cervical vertebra. Its cavity communicated freely with the esophagus. A little ribbon of opaque fluid passed down from this diverticulum to the opening on the skin.

The author thinks the diverticulum was not caused by the foreign body but that it was a Zenker's pulsion diverticulum which had existed before the bone was swallowed. The bone or other food lodged in the diverticulum probably caused inflammation and finally abscess formation and the pus burrowed down until the fistula was formed.—*Audrey G. Morgan.*

Torelli, Gastone. Contributo allo studio del torace normale; l'ombra satellite sottoclaveare. (Study of the normal thorax; the satellite subclavicular shadow.) *Radiol. med.*, July, 1940, *27*, 576–578.

It is very important to know the roentgen picture of the normal thorax, as otherwise pathological findings cannot be interpreted. It

has long been known that there is a shadow above the clavicle in normal subjects when the supraclavicular fossa is very deep. The author now describes and illustrates a similar shadow below the outer end of the clavicle, caused also by soft parts and showing when the subclavicular fossa is unusually pronounced. He shows a roentgenogram illustrating this subclavicular shadow, and then another of the same subject with the shoulder in abduction. In this roentgenogram the subclavicular shadow has disappeared, because the abduction of the shoulder has reduced the depth of the subclavicular fossa.—*Audrey G. Morgan.*

Hill, Frederick T. Diagnostic pitfalls of the nasopharynx. *J. Am. M. Ass.*, Aug. 31, 1940, *115*, 662–665.

The main pitfalls in the diagnosis of nasopharyngeal disease might be considered as due to four causes: (1) not looking; (2) not seeing; (3) not knowing what to look for; (4) not interpreting correctly what is seen.

Unless prevented by marked septal deviation, the nasopharyngoscope should be passed through both sides of the nose so that the blind area directly ahead of the instrument can be included. By direct inspection, using either the speculum or a retractor, one can see the actual picture without distortion and can better interpret the condition seen with the nasopharyngoscope or the mirror. The roentgenogram should be employed in all cases of suspected malignant disease.

As common a pitfall as not accurately seeing the nasopharynx is not knowing what to look for. Among the things which may be found are adenoids, hypertrophy of the posterior tips of the inferior turbinate or adhesions across the fossa of Rosenmüller or the eustachian orifice, postnasal discharge which may indicate a chronic posterior sinusitis, choanal polypi or the more serious nasopharyngeal fibromas, tumors developing from the remnants of the hypophysial duct, classified by Furstenberg as craniopharyngiomas, chordomas arising from the fetal notochord, nasopharyngeal cysts or bursae, primary tuberculosis, and congenital defects, such as atresia of the posterior choanae. Suppuration of the petrous apex may point in the nasopharynx.

Malignant tumors of the nasopharynx often manifest themselves first by extra-nasopharyngeal signs and symptoms, usually through involvement of the cranial nerves. New has emphasized the diagnostic importance of diplopia, facial pain and disturbances in swallowing as early symptoms. The cranial nerves most commonly involved are the sixth, fifth, ninth, tenth, eleventh and twelfth. The presence of posterior cervical adenopathy, pain referred to the throat or ear and signs of a unilateral conduction deafness may indicate a malignant growth of the nasopharynx.—*S. G. Henderson.*

Figi, Frederick A. Fibromas of the nasopharynx. *J. Am. M. Ass.*, Aug. 31, 1940, *115*, 665–672.

Juvenile basal fibromas of the nasopharynx are rare; few reports in the literature present more than a half dozen cases seen by a single individual. These tumors occur predominantly among male patients. They arise most commonly in the vault or high on the posterior wall of the nasopharynx from the periosteum covering the basilar process of the occipital bone and the body of the sphenoid bone. They appear at about the age of puberty and continue to be active until the age of approximately twenty to twenty-five years, at which time they tend to regress spontaneously and in some instances disappear completely. Fibromas of the nasopharynx usually appear as hard, often almost cartilaginous, rounded, slightly nodular or lobulated, deep red, purplish or grayish tumors occupying the upper portion of the nasopharynx and often extending into one or both nasal fossae as well. Prolongations are said to grow most intensively where they encounter the greatest obstacles. The tumor is covered with normal mucous membrane, and tortuous vessels often stand out prominently on the surfaces.

Pressure of the expanding neoplasm often results in a remarkable degree of destruction of the bone with which it comes in contact. Nasal obstruction was the first symptom noted by most of the patients. Pain was not a prominent feature in any of the cases.

Roentgenographic studies will often furnish considerable information additional to that obtainable on physical examination alone. The tumor in the nasopharynx will, as a rule, be revealed best in lateral roentgenograms. Roentgenograms in 40 of 45 cases showed evidence of pathologic processes in the sinuses, nose, nasopharynx or orbit in 33 individuals. The pathologic changes included the presence of a tumor mass in one or more of the sinuses or in the adjacent structures, diffuse dullness of a single

sinus, pansinusitis and, in a few instances, only thickened membrane. These tumors are primarily pure fibromas and microscopically consist of dense connective tissue. They are extremely vascular and the vessels are often cavernous in type. In addition, more or less inflammatory reaction is usually present. Cellular portions commonly are present in the tumors and often lead to a mistaken diagnosis of fibrosarcoma. The chief objection to removal of tissue for histopathologic study is the profuse bleeding that invariably ensues after such a procedure. At times this bleeding is difficult to control and, if positive recognition is possible without microscopic examination, biopsy should be avoided. In the literature many types of tumors appear to have been confused with fibromas of the nasopharynx; among these, myxomatous choanal polyps which had attained considerable size, and also periosteal fibromas, fibrosarcomas and various other types of malignant neoplasms.

The treatment of nasopharyngeal fibromas has undergone considerable change. Prior to 1915 surgical procedures were resorted to almost exclusively. Experience generally with such measures was a high mortality rate and frequent recurrence, necessitating repeated operations. At the Mayo Clinic subsequent to the beginning of 1919, radium therapy or electrocoagulation, either alone or combined, was employed in the treatment of 42 patients. Since the beginning of 1924 surgical diathermy has been used with increasing frequency in the treatment of these tumors, and in most instances it has been supplemented with radium. The one patient for whom roentgen therapy was used was a hemophiliac. With the exception of 2 instances, the average length of time the patients receiving diathermy and radium combined were under treatment was ten months. Fewer complications are likely to be encountered if treatment is carried out in stages, rather than at a single sitting.

There has been no mortality in these cases to date and all the patients who have received the therapy either have been cured or are well on their way to complete recovery.—*S. G. Henderson.*

Walsh, Frank B., and Goldberg, H. Krieger. Blindness due to air embolism: a complication of extrapleural pneumolysis. *J. Am. M. Ass.*, Feb. 24, 1940, *114*, 654–655.

The authors wish to draw attention to the occurrence of an air embolus in the eye and to record 2 cases in which this was observed as a complication of extrapleural pneumolysis. Both patients were blind for a period of three days. Thereafter vision gradually improved and returned to normal by the end of two weeks.

The mechanism of air embolism is not entirely clear. It is probable that prerequisites for the occurrence of air embolism are open or wounded veins, available air and either pressure on the available air so that it is forced into the veins or a negative pressure within the veins so that the air is pulled into them.

The occurrence of symptoms usually follows immediately after the injection of air, but in some instances there may be a lapse of several hours between the injection of air and the onset of symptoms. The patient complains immediately of severe pains in the head, dizziness, nausea and vomiting. In some instances there may be blindness. Fear of impending death is a prominent symptom. The pulse becomes rapid and the breathing shallow and labored. There may be general or localized convulsions. Extensive paralyses may develop. Death may ensue within a few minutes. If death does not occur within the first half hour the prognosis is relatively good.—*S. G. Henderson.*

Ellison, Richard T., and Carabelli, A. Albert. Tense pneumothorax in an infant; successful treatment by the production of chemical pleuritis. *Am. J. Dis. Child.*, Sept., 1940, *60*, 644–651.

The condition in infants and children known as the so-called valvular or tense pneumothorax is rarely reported in the literature. The authors report a case occurring in an infant four and one-half months old. On admission to the hospital, a roentgenogram showed a large pneumothorax in the left side of the chest, with adhesions binding the lung to the lower part of the wall of the chest and to the diaphragm.

Valvular or tense pneumothorax is the type in which intrapleural pressure remains above atmospheric pressure in both phases of quiet respiration It is the result of a one way valve action through a rent in the visceral pleura. Forceful inspiratory effort, as in crying, pumps enough air into the pleural space to exert a positive pressure during the respiratory phase of quiet breathing. This trapped air gradually reduces the vital capacity, which in turn stimulates greater respiratory effort, and a vicious

circle results. If the mediastinum is rigid, effects of pressure are felt only on the involved side, and the contralateral lung can function normally and supply gas exchange adequate for quiet activity. If the mediastinum is mobile, however, the trapped air pushes it into the contralateral hemithorax, gradually compressing the healthy lung until the patient dies of suffocation.

In regard to the vent in the visceral pleura which results in spontaneous pneumothorax, cases fall into three groups: (1) those due to direct trauma; (2) those in which a necrotizing lesion of the lung has destroyed the continuity of the pleura before obliterative pleurisy has developed; (3) those in which the pneumothorax results from localized overinflation of the pulmonary parenchyma, which causes bursting of the alveoli and overlying pleura.

The authors treated their patient successfully by induction of chemical pleuritis with a solution of 50 per cent solution of dextrose.—*R. S. Bromer.*

Caffey, John. Regional obstructive pulmonary emphysema in infants and in children; emphysematous cavities and their similarity to necrotic cavities, congenital pulmonary cysts and loculated pneumothorax. *Am. J. Dis. Child.*, Sept., 1940, *60*, 586–605.

Caffey reports the clinical and roentgenological observations on 7 infants and children in whom pulmonary cavities developed during infections of the respiratory tract. In the differential diagnosis the following lesions were considered as possible causes of the cavities seen in the roentgenograms: (1) acute pulmonary abscess, (2) aputrid pulmonary necrosis, (3) tuberculous cavitation, (4) loculated pneumothorax or pyopneumothorax, (5) congenital cysts of the lung and (6) regional obstructive emphysema.

Caffey discusses in detail the differential diagnosis and concludes that in his cases the clinical and serial roentgenographic features were consistent with regional obstructive emphysema and were not consistent with pulmonary abscess or congenital cystic disease of the lung. He raises the question whether air-fluid levels in pulmonary cavities are pathognomonic of pulmonary abscess and suggests that in these cases inflammatory fluid exudate in emphysematous cavities caused shifting air-fluid levels similar to those produced by necrotic fluid in necrotic cavities. The literature is reviewed dealing with the morbid anatomy, the roentgenographic signs and the bronchoscopic observations in regional emphysema and with its causal mechanism, incomplete bronchial obstruction.—*R. S. Bromer.*

Sailer, Seaton. So-called epituberculosis. *Am. J. Dis. Child.*, Oct., 1940, *60*, 900–906.

Sailer reports a case of boy, four years of age when first seen, in whom a large area of pulmonary atelectasis persisted for three years. The patient exhibited only mild clinical symptoms and had a strongly positive tuberculin reaction. When the atelectatic area cleared, prominent lymph node shadows were demonstrable at the hilum of the involved area. The massive pulmonary shadows noted on the roentgen examination appeared to be caused by resorption atelectasis consequent on compression of the bronchus by enlarged tuberculous lymph nodes.

Sailer thinks the epituberculous lesion occurring in children is constantly associated with the complex of primary tuberculosis of the lymph nodes. Various pathologic changes may result from involvement of the bronchial lymph nodes in the primary tuberculous lesion, and he suggests that the term epituberculosis be confined to those cases in which pulmonary atelectasis resulting from bronchial compression is followed by resolution and clinical recovery.—*R. S. Bromer.*

Fossati, Franco, and Valsecchi, Romeo. Quadri radiologici della polmonite crupale. (Roentgen findings in lobar pneumonia.) *Radiol. med.*, July, 1940, *27*, 525–550.

The authors have made a roentgen study of 90 cases of lobar pneumonia, making the examinations in all the stages of the disease. Thirty-eight typical roentgenograms are reproduced.

They have paid special attention to certain points that have not been very carefully studied before, such as the localization of the focus in the beginning, the relation between the time of appearance of the clinical and roentgen signs of the disease and the frequent limitation of hepatization to certain peripheral areas which have some anatomical and functional autonomy. These areas were called zones by Pierret; they are quite constantly distributed in different patients and a diagrammatic drawing of their localization is given.

Roentgen examination in pneumonia does

not involve the slightest danger to the patient if the proper precautions are taken. If the patient is in very serious general condition it may be well to take a portable roentgen apparatus to the bedside rather than to transport the patient to the roentgen room.

Roentgen examination is useful in all stages of the disease. In the stage of congestion it may show changes in the parenchyma before they are demonstrable on physical examination. In the stage of hepatization the exact location and extent of the pneumonic foci can be determined. In the stage of regression the clearing up of the shadow can be followed up and any complications detected. In the stage of resolution the clearing of the shadow and the reduction in size of the foci of pneumonic consolidation begin at the same time in all the peripheral foci and progress toward the center. Generally complete disappearance of the shadow takes place within from ten to fifty days. In cases of delayed resolution differential diagnosis from other chronic inflammations of the lung, particularly tuberculosis, is very difficult and requires a long series of roentgen examinations.

For accurate localization of the pneumonic foci it is is necessary to make examinations not only in the sagittal projection but also in lateral and oblique projections.

It is rarely possible to determine the exact site of the beginning of the process by roentgen examination. The findings of foci in perihilar or parahilar localizations which do not diffuse to the periphery until later does not argue in favor of the bronchogenic origin of lobar pneumonia. It has been shown that certain of these "central" foci have been projected to the hilar region but are really localized immediately beneath the mediastinal or interlobar pleura and are to be considered cortical foci. In the stage of congestion the roentgen signs are clearly evident; so it cannot be said that the clinical signs appear before the roentgen signs.

In the transition stage between congestion and hepatization the foci sometimes increase very rapidly, which is shown by increased extent and density of the roentgen shadow. In the later course of the disease the foci do not change much. In the stationary period of the disease the shadows vary in location and extent but they have no pathognomonic characteristics. Differential diagnosis from other diseases of the lung, such as bronchopneumonia and tuberculosis is difficult and often impossible; it must be based on clinical signs and the course of the disease rather than on roentgen examination.—*Audrey G. Morgan.*

Editorial. The superior pulmonary sulcus tumor of Pancoast. *J. Am. M. Ass.*, Aug. 24, 1940, *115*, 617–618.

In 1924 Pancoast reported 3 cases of neoplasms occurring at a definite location at the thoracic inlet, characterized by pain around the shoulder and down the arm, Horner's syndrome and atrophy of the muscles of the hand, roentgenologic evidences of a homogeneous shadow at the apex, always more or less local rib destruction and often vertebral infiltration. The name "superior pulmonary sulcus tumor" was given to this new growth indicating its approximate location and the "lack of origin from the lung, pleura, ribs or mediastinum." A biopsy in one of Pancoast's cases revealed an epithelioid carcinoma. Pancoast believed that the tumor had its origin from an embryonic remnant of the fifth branchial pouch.

Since these cases were reported there has been much controversy regarding the specificity of both the syndrome and the tumor, as well as its origin as suggested by Pancoast.

Recently Morris and Harken in an analytical review of the literature on the subject and 8 personal cases concluded that the Pancoast tumor is a specific entity. They believe that the Pancoast syndrome can be initiated by a variety of conditions, but that the Pancoast tumor is a definite pathologic entity. They insist on certain criteria in making the diagnosis: clinical evidence of an apical tumor, histologic evidence of an epithelioma and pathologic evidence based on postmortem studies which are competent to prove the extrapulmonary character of the tumor, its lack of origin from lung, pleura, ribs or mediastinum and its primary nature.

Certain clinical features about the Pancoast syndrome which may be emphasized as characteristic are the onset with excruciating pain about the shoulder and the apex of the axilla, the constant and early development of the Horner syndrome, and the constant invasion of the first, second and third ribs and of the vertebrae. The tumor is malignant and refractive to irradiation. Surgical removal is difficult.—*S. G. Henderson.*

Calchi-Novati, Giulio. Cisti unica congenita del polmone. (Solitary congenital cysts of the lung.) *Radiol. med.*, July, 1940, *27*, 556–576.

The various theories in regard to the formation of congenital cysts of the lung are discussed; none of them has been definitely proved. These are not true cysts in the ordinary sense of the word as the sac may not be closed and they are generally filled with air. In the adult it is often difficult to decide whether a cyst is congenital or acquired; only those found in newborn or stillborn infants are definitely known to be congenital.

These cysts may persist for years without causing any symptoms and become clinically manifest only after infection has taken place. In infants, if the cyst is very large, it may cause symptoms similar to those of suffocating pneumothorax. In adults it may be complicated by hemoptysis and infection.

A case is described in a woman fifty-nine years of age admitted to the hospital with signs of heart failure and with chronic bronchitis, probably bronchiectatic. She had had good health up to the age of thirty-nine, when she had an attack of bronchopneumonia. From that time on she had recurrent attacks of bronchitic and pulmonary infection and finally developed signs of heart disease. Roentgen examination showed a cavity at the base of the right lung clearly outlined by an almost exactly circular ring; its lower edge passed into the liver-diaphragm dullness. After excluding the various forms of acquired cysts, tumor and tuberculous cavity, the author concluded that this was a true congenital cyst which for many years had caused no symptoms.

The author emphasizes the importance of roentgen examination in demonstrating these cysts, and not merely demonstrating the cyst as such, which is of value as these cysts can often not be diagnosed clinically, but in differentiating them from the other similar pathological processes which are more or less frequently found in lung tissue.—*Audrey G. Morgan.*

Sosman, Merrill C. Subclinical mitral disease. *J. Am. M. Ass.*, Sept. 28, 1940, *115*, 1061–1066.

Mitral stenosis is almost invariably acquired and is practically always the result of rheumatic fever. When relative, mitral insufficiency is secondary to dilatation of the left ventricle, but organic mitral insufficiency is almost always the direct result of rheumatic fever. Dilatation of the left auricle will almost invariably ensue in both conditions. Therefore, careful roentgen study may be of help in the diagnosis of mitral disease by demonstrating the dilated left auricle. If stenosis is associated with mitral insufficiency, there will be enlargement of the left ventricle.

The diagnosis of mitral stenosis can be made by auscultation, roentgen examination and electrocardiography. In the early stages on auscultation there is a snapping first sound and a short pre-systolic or mid-diastolic rumbling murmur. In more advanced degrees of mitral stenosis the murmur may fill all of diastole. The next most important method of confirming the diagnosis is the demonstration of the dilated left auricle roentgenoscopically or roentgenographically. The third observation of critical importance is the presence of a broad, flattened P wave in the electrocardiogram, especially if right axis deviation is also present.

On roentgen examination dilatation of the left auricle is best seen in the right anterior oblique position. In the posteroanterior view the change in the cardiac contour may be less marked and less characteristic. There may be only a straightening of the normally concave "waist" of the heart on the left border, a contour not typical of mitral disease. In the more advanced cases the left contour presents the double convexity of the dilated pulmonary artery and tip of the dilated left auricle between the aortic knob and the left ventricular contour.

Roentgenoscopic examination is much more valuable in the study of heart disease than are roentgen films. It is the only reliable method of finding, locating and identifying areas of intracardiac calcification during life. The roentgenoscopic examination of a patient with mitral valvular disease should reveal: 1. A straightening or a double convexity of the midportion of the left cardiac contour. 2. Dilatation of the left auricle posteriorly. 3. Widening of the base of the heart in the right anterior oblique view. There may also be seen: 4. Displacement of the barium-filled esophagus posteriorly. 5. Dilatation of the pulmonary vessels at the lung roots. 6. Hypertrophy of the right ventricle. 7. Narrowing of the precardiac space in the lateral view. 8. A hypoplastic aortic knob, in cases in which the mitral stenosis was acquired in childhood. In advanced or long standing mitral valvular disease, the left auricle may be so dilated that it will displace the left bronchus upward and may even compress it. This may result in dyspnea, cough, atelectasis or obstructive emphysema of the left upper lobe.

In the author's experience, the demonstration of calcified areas in the mitral valve is absolutely pathognomonic of mitral valvular disease, but calcification in the injured mitral valve is not visible roentgenoscopically in more than 10 per cent of the patients during life. These intracardiac deposits of calcification are most easily located roentgenoscopically. The observer's eyes must be thoroughly prepared by at least fifteen minutes in a completely darkened room. The deposits all show a to and fro movement with systole and diastole. The calcified aortic and mitral valves are found under a line along the auriculoventricular sulcus, which is usually about 45 degrees from the horizontal. The patient should be rotated slightly to the left so that the calcified areas will not be hidden by the spine. The patient should take a deep inspiration and hold it, so that the underlying pulmonary markings will be immobile and not confusing. A reliable means of differentiating between calcified aortic and mitral valves is to rotate the patient to the opposite side (left anterior oblique), where the mitral valve will be found to be in the posterior third of the heart shadow, while the aortic valve will be found in the middle third. A frequent source of error is calcification of the mitral annulus fibrosus. The calcified annulus is usually either U shaped or J shaped or O shaped, and it is more homogeneous than the irregular mulberry-like calcification in the valves themselves. It occurs only as a senile change, usually in persons over sixty years of age. The author believes that the presence of calcification in the mitral valve indicates rheumatic etiology, as he has not found this degree of calcification in the mitral valve to be due to either syphilitic or atherosclerotic processes.

By "subclinical mitral disease" is meant patients with mitral disease whose condition was clinically undiagnosed before roentgen examination. These have been termed class A, B and C. In the class A group these pateints have unequivocal roentgen signs of mitral disease, but the clinical observations are either absent or inconclusive. Class B is the largest group and includes the patients with undoubted mitral disease in whom the physical signs of the disease were overlooked but in whom the appearances by roentgenoscopic and film examinations were quite definite. Class C includes those patients with the roentgen findings compatible with mitral disease but not characteristic of it and with no clinical or other observations to corroborate the diagnosis. The most frequent cause of error in the diagnosis of mitral disease is in heart failure from any cause, particularly with cardiac dilatation and auricular fibrillation. The dilated left auricle seen in these conditions may actually be due to relative mitral regurgitation or to stasis in the atonic fibrillating auricle.

Accuracy in the early stages of mitral disease is open to question, as there is often a long latent period between a given attack of rheumatic fever and the discovery of mitral stenosis. The process must inevitably have been present and gradually progressing during the latent period when no murmurs, or only a systolic murmur, were present. Mitral stenosis does not appear suddenly; it is a slow gradual process. It is quite possible, therefore, that the routine use of the roentgenoscope by a trained observer may be of aid in the earlier diagnosis of mitral stenosis.

Several illustrative case histories are included in this article.—*S. G. Henderson.*

ABDOMEN

PIEROSE, PERRY N. Hemangioma of the gastrointestinal tract. *J. Am. M. Ass.*, July 20, 1940, *115*, 209–211.

The author reports one case in which hemangioma of the gastrointestinal tract was proved and a review of the literature concerning this condition. This patient's symptoms were of fifteen years' duration and were severe from the onset. The first symptom of her present condition came on rather suddenly in 1926, when she was twenty-nine years of age, and consisted of great weakness. This was followed by pallor, tarry stools and "extreme anemia." The symptoms developed suddenly, and subsided while the patient rested in bed at home. Following this experience she was admitted to the Los Angeles County Hospital six times during the next thirteen years. Following each of these episodes the patient's condition would improve, apparently as a result of bed rest, transfusions with whole blood, a well balanced, adequate diet and administration of iron. Exploratory laparotomy was done in 1932 and again in 1933, but the cause of her symptoms was not found. By roentgen examination of the gastrointestinal tract the duodenum was considered to be slightly enlarged, passage of barium sulfate through it was somewhat delayed, and it was suggested that these changes might result from the presence of a duodenal polyp. An enema of barium

sulfate revealed no abnormalities of the colon. In 1932 exploratory laparotomy was performed and a peculiar band was found along the antemesenteric border of the jejunum, which produced in it an appearance similar to that seen in the large intestine. Sacculations containing enlarged blood vessels and engorgement of the smaller vessels were found in the lateral walls of the jejunum. At this operation and at operation on April 10, 1933, the cause of hemorrhage from the bowel was not found.

Laparotomy was again performed March 1, 1939. The increased vascularity of the jejunum was again observed. Telangiectases were present in the serosa of the jejunum and the proximal portion of the ileum. Three feet of small bowel extending from 1 foot distal to the origin of the jejunum downward were removed and an end to end anastomosis made. By microscopic examination of numerous sections of the jejunum, increased vascularity was seen in all layers. Large spaces lined with endothelial cells and containing blood were found in the serosa and muscularis. Occasional plexiform groups of small thin walled vessels were also present. The pathologic diagnosis was diffuse telangiectatic cavernous hemangioma of the jejunum.

The patient has been free from symptoms and leading a normal life for a period of more than nine months.

When a patient experiences repeated attacks of melena which cannot be explained by a thorough study of the history, physical manifestations and laboratory examinations, the possibility of hemangioma of the gastrointestinal tract should always be considered.—*S. G. Henderson.*

Williams, Carrington, and Kimmelstiel, Paul. Syphilis of the stomach. *J. Am. M. Ass.*, Aug. 24, 1940, *115*, 578–582.

In syphilis of the stomach the lesion is an infiltrating one which involves varying amounts of the stomach and is located primarily in the submucosa. Ulceration is secondary and therefore usually shallow. In the great majority of cases the lesion is located in the pylorus. The next most frequent location is the mid-stomach, where an hour-glass deformity is produced. A leather-bottle deformity may be produced in syphilis of long standing.

The diagnosis can positively be made only after long continued successful antisyphilitic therapy or by the gross appearance and microscopic picture of removed tissue. Pain, weight loss and vomiting are prominent symptoms. A positive Wassermann reaction, achlorhydria, or low free and total acid and secondary anemia are the usual findings. The roentgen appearance is the most important diagnostic sign. Most authors say that this appearance is indistinguishable from cancer, but Williams in 1935 emphasized the smooth tube-like or funnel deformity as characteristic of syphilis.

The gross appearance of the lesion is much less extensive than the roentgen appearance. In early cases the stomach is pliable. Later it has a tough but smooth feel.

Histologic sections reveal a more or less diffuse infiltration of the entire wall with a variety of round cells, predominantly lymphocytes and plasma cells. The infiltration is most marked in the submucosa. A conspicuous perivascular arrangement of this inflammatory infiltration is noticeable. Proliferating endarteritis is a common finding but panphlebitis is more often found than endarteritis, although it can be detected only with special stains for elastic fibers. The phlebitis, as one of the outstanding criteria in aid for differential diagnosis, has not found the attention it deserves. The authors refer to this type of panphlebitis as periendophlebitis, indicating the direction of progress of the granulation tissue penetrating the wall of veins from outside in, thus distinguishing it from other types of phlebitis primarily beginning in the vascular coats proper. In tertiary syphilis this type of venous destruction seems to occur with particular frequency in the gastrointestinal tract. Periendophlebitis is occasionally noticed in other types of specific infiltrating granulation tissues (particularly venereal lymphogranuloma).

The diagnosis of syphilis of the stomach was definitely proved by Harris and Morgan, who produced a chancre of the testicle in a rabbit by injecting macerated tissue from one of their patients. From this chancre spirochetes were recovered which in turn produced another similar lesion.

Medical treatment is indicated in early cases when the diagnosis is reasonably certain and when complications have not appeared. In the later cases improvement may occur on medical treatment but it is far from cure. In the differential diagnosis between syphilis and cancer the diagnosis of syphilis is suggested by the funnel or tube-like deformity and other observations,

such as a positive Wassermann reaction, a younger patient and a fairly good general condition. The response to antisyphilitic therapy is of little value because almost any stomach lesion will improve during treatment. In spite of adequate therapy the deformity is only moderately changed. In cases of marked deformity resection of the stomach should be done. When complications exist such as hemorrhage, pyloric obstruction, disabling pain and marked weight loss it is useless to delay operation. In 6 of the authors' 9 cases the pyloric end of the stomach containing the involved area was resected. All of these patients survived and were rapidly restored to health.—*S. G. Henderson.*

BORIANI, GIUSEPPE. Anatomo-radiografia dell' ulcera duodenale. (Anatomo-roentgenography of duodenal ulcer.) *Radiol. med.*, July, 1940, *27*, 551–556.

The author made what he calls anatomo-roentgenographic examinations of 10 cases of duodenal ulcer, that is he filled the resected surgical specimens with contrast medium and made roentgenograms of them. This gives quite a different picture from that obtained by roentgenography of the living subject. Descriptions, illustrated with roentgenograms, are given of the clinical and roentgen findings before operation and the roentgen findings in the resected specimens.

It was found that the ulcer niche is practically always in the center of the anterior or posterior wall. Though the roentgen findings before operation had shown the niches on the lesser curvature in 7 of the cases, the anatomical roentgenogram showed them all on the anterior or posterior wall. It has been held that the ulcer may begin on the wall and extend later to the lesser curvature, and one of the cases seemed to indicate that this may be true. There were "spines" running out from the niche which were folds in the mucous membrane, incomplete in the direction of the curvature because they had encountered the curvature; but the ulcer itself was on the wall. The folds always converge in the anatomical specimen, though it may be impossible to demonstrate this on the living subject. The bulb of the duodenum may or may not present the anatomical retractions and diverticula described by Åkerlund; they are probably less frequent than is generally believed.—*Audrey G. Morgan.*

FANTUS, BERNARD, KOPSTEIN, GEZA, and SCHMIDT, HILMAR R. Roentgen study of intestinal motility as influenced by bran. *J. Am. M. Ass.*, Feb. 3, 1940, *114*, 404–408.

Authors' Conclusions. Studies as to the influence of bran on emptying time of the bowel in normal individuals may be summarized as follows:

1. (a) Acceleration of the optimal cecal emptying time (twenty-four hours) is evident in but few cases.
   (b) Bran accelerates the emptying time in most cases which show a forty-eight hour cecal emptying time.
2. The daily use of bran decreases the total emptying time of about 25 per cent of normal persons. This decrease varies from twenty-four to sixty hours.
3. Bran produces variations in the tonus of the colon. In cases in which hypertonic haustrations are present, a marked reduction of the spasm has been noted after the use of bran.
4. The appendix was visualized in some cases after bran ingestion in which control observations did not show that organ. This makes it desirable to make further special studies of the effect of bran on the poorly draining appendix. Observation invites a close study of the uses of bran in cases of spastic colon. It is essential to determine in which cases of spastic colon it may be used as a preventive of paroxysms of colic and in which types it should not be used at all.—*S. G. Henderson.*